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PRIMARY INTRATHORACIC NEUROGENIC TUMORS

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INTRODUCTION

THE purpose of this paper is to present a clinicopathologic study of 24 cases of primary intrathoracic tumors of nerve origin with special emphasis on the classification and pathologic features of these tumors.

REVIEW OF LITERATURE

In 1944, Kent and associates[1] in a paper entitled "Intrathoracic Neurogenic Tumors" collected 105 cases from the literature and added 21 new cases, 18 of which were acceptable histologically. According to the documentation presented, about 78 of the collected cases were actually primary intrathoracic neurogenic lesions. Twelve (15.3 per cent) of the 78 were malignant in contrast to the figure of 37 per cent for the entire series. Of the 18 new cases 41 per cent were classified as malignant. Blades,[2] in 1946, reported 1 neurosarcoma (3.3 per cent) among 30 mediastinal neurogenic tumors. Curreri and Gale[3] stated that 92 per cent of posterior mediastinal tumors are neurogenic and they reported 6 benign and 1 malignant (14.3 per cent) primary nerve tumors in a series of 36 mediastinal tumors. Brewer and Dolley[4] reported 16 benign and 1 malignant neurogenic tumors in a series of 44 mediastinal tumors.

Harrington[5] recorded 51 primary nerve tumors of the posterior mediastinum, of which 5.9 per cent were malignant. Arbuckle[6] reported 11 primary intrathoracic nerve lesions, 2 (18.1 per cent) of which were malignant. Rarely, primary neurogenic lesions are recorded of the anterior mediastinum,[1] lung,[7, 8] diaphragm,[9] and lateral chest wall.[8] Several of the reported cases of medias-

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TABLE I

CASE	SEX	AGE (YRS.)	SYMPTOMS	LOCATION	GROSS PATHOLOGY	SURGERY		FOLLOW-UP
Case of Diffuse Neurilemoma								
1. C. F.	M	22	Hemoptysis, pain	Anterior mediastinum	Tissue for biopsy	2-40	Biopsy	Death from infected, fungating tumor, 1-46
Cases of Encapsulated Neurilemoma								
2. D. M.	M	26	Asymptomatic	Right upper posterior mediastinum	Encapsulated, firm, 3.5 cm. in diameter; cut surface lobulated, golden yellow to gray	10-42	Total excision	11-45 Asymptomatic, negative x-ray
3. P. S.	M	25	Chronic cough	Left upper posterior mediastinum	Not available; submitted slide	1943	Total excision	9-49 Asymptomatic
4. S. S.	F	36	Asymptomatic	Right lower posterior mediastinum	Encapsulated, cystic tumor with necrotic center, 10 cm. in diameter; lining of soft, brown, friable tissue with yellow areas	4-44	Total excision	7-48 Asymptomatic
5. N. C.	M	50	Fatigue	Right posterior inferior mediastinum	Encapsulated, cystic tumor, 7.5 by 6 by 3 cm. in diameter; on section, two-thirds of tissue composed of blood filled cysts, surrounding tissue soft and yellow brown	6-45	Total excision	9-49 Asthmatic, negative x-ray
6. P. B.	F	31	Pain, right chest for one year	Right upper posterior mediastinum	Encapsulated, firm tumor 3.5 by 5 cm. in diameter; cut surface yellow to gray	9-46	Total excision	9-49 Asymptomatic
7. K. R.	M	25	Cough for one year	Right upper posterolateral chest wall; origin in third intercostal nerve (intrathoracic)	Encapsulated, firm tumor 3 by 2.5 by 2.5 cm.; cut surface homogenously grayish-white	10-46	Total excision	1948 Asymptomatic, negative-x-ray
8. J. S.	M	35	Asymptomatic	Right upper posterior mediastinum	Encapsulated, cystic tumor 4 by 3 by 2 cm.; cut surface composed of two thin-walled, blood-filled cysts	1-47	Total excision	None

9.	S. L.	M	18	Asymptomatic	Right lower posterior mediastinum	Encapsulated, reddish-brown, 3 by 3.5 by 2 cm.; periphery of cut surface firm, reddish-brown with patulent vessels in center	6-47	Total excision	None	
10.	M. B.	F	57	Dyspnea (left pleural effusion)	Left upper posterior mediastinum	Encapsulated, cystic tumor 15 by 12 by 9 cm.; section showed blood-filled cysts and yellow to gray tissue; some areas appeared necrotic	9-47	Total excision	8-49	Asymptomatic, negative-x-ray
11.	E. F.	F	37	Pain, right shoulder for two years	Right upper posterior chest wall; origin in second intercostal nerve (intrathoracic)	Encapsulated, firm orange-tan, largely solid except for a few small blood-filled cysts. 7 cm. in diameter	4-49	Total excision	None	
12.	C. F.	F	30	Asymptomatic	Left posterior mediastinum	Encapsulated, firm tumor 9 by 7 by 6 cm. weighing 180 grams; cut surface was firm, pale gray, streaked with yellow-orange zones	11-49	Total excision	1-50	Asymptomatic, negative-x-ray
13.	I. B.	F	35	Asymptomatic	Left upper posterior mediastinum	Encapsulated, firm tumor 3 by 2.5 by 1.5 cm.; cut surface firm, bright yellow to gray	3-50	Total excision	4-50	Asymptomatic, negative-x-ray
					Case of Malignant Neurilemoma					
14.	L. G.	F	13	Cough for ten years (diffuse neurilemomatosis)	Right posterior mediastinum	Four encapsulated, hard masses 8 by 7 by 4 cm., 4 by 3 by 2 cm., 2 by 2, and 1 by 2 cm.; cut surface white with yellow streaks and mucoid areas	11-44	Partial excision	7-45 11-45	Local recurrence Death (cachexia)
					Cases of Ganglioneuroma					
15.	A. P.	M	26	Left chest pain for three months	Left upper posterior mediastinum and surrounding subclavian artery	Encapsulated, firm tumor 5 cm. in diameter; cut surface firm, smooth, moist, and light tan	3-45	Excision of thoracic portion; tumor surrounding subclavian artery not removed	7-49	Asymptomatic, negative x-ray; no palpable tumor in neck
16.	T. H.	M	19	Asymptomatic	Right lower posterolateral chest wall (intrathoracic)	No. 1 encapsulated, slightly nodular solid, firm, pearly-white to yellow tumor, 2 by 6 by 13 cm. in diameter	10-46	No. 1, specimen partial excision	1-49	Asymptomatic, negative x-ray

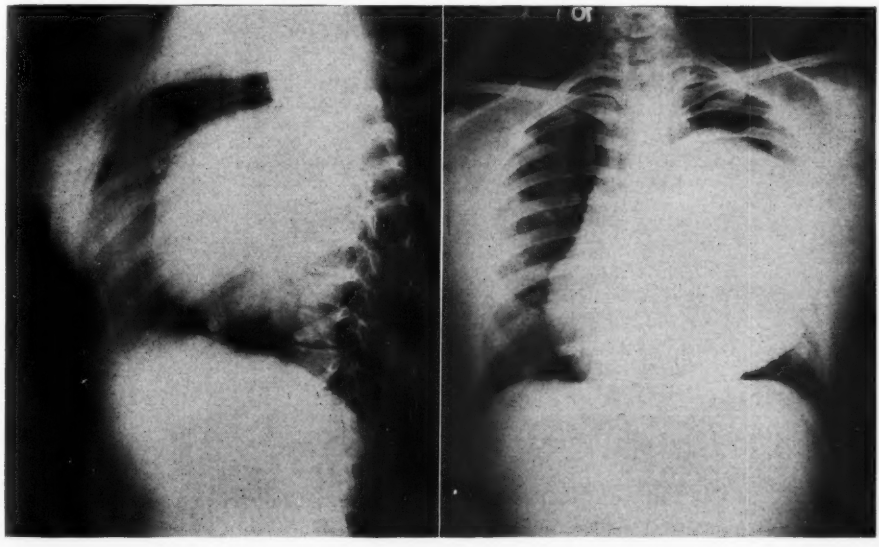
TABLE I—CONT'D

CASE	SEX	AGE (YRS.)	SYMPTOMS	LOCATION	GROSS PATHOLOGY	SURGERY		FOLLOW-UP	
17. R. H.	F	46	Asymptomatic	Right posterior mediastinum	No. 2, encapsulated, firm yellow-gray, translucent, solid tumor 14 by 12 by 4.5 cm. in diameter	4-47	No. 2, specimen total excision of remaining tumor		
					Encapsulated, firm homogenous, glistening, grayish-yellow with attached nerve trunks; 10 by 7 cm. in diameter	11-46	Total excision	1-50	Asymptomatic, negative-x-ray
18. N. F.	F	5	Asymptomatic (café-au-lait spots)	Left and right, upper posterior mediastinum and within left brachial plexus	Tissue for biopsy	5-47	Left thoracotomy, biopsy	1-49	Asymptomatic, x-rays unchanged
19. R. O.	F	6	Cough and dyspnea for one year (identical twin without disease)	Right and left posterior mediastinum	Encapsulated, firm 14 by 10 by 3 cm.; cut surface solid, grayish-white; few attached nerves	7-47	Excision complete except for right intercostal and left posterior mediastinal extensions	9-49	No recurrence at operative site; no symptoms suggesting progression of residual tumor
								3-50	Asymptomatic except for asthma
20. M. E.	F	14	Dyspnea	Left posterior mediastinum	Three masses together weighing 1,150 grams, incomplete thin capsule; soft, yellow with several attached nerves	11-48	Total excision	5-49	Asymptomatic, x-ray negative
21. J. M.	F	9	Cough	Right and left posterior mediastinum and retroperitoneal	Tissue for biopsy	1-49	Left thoracotomy, biopsy	3-49	Asymptomatic, x-ray unchanged
22. J. H.	F	14	Asymptomatic	Left posterior mediastinum	Encapsulated, soft homogenous, yellowish-gray tumor 2 by 6 by 13 cm. in diameter	8-49	Total excision	1-50	Asymptomatic, negative x-ray
Case of Sympathicoblastoma									
23. J. E.	F	7	Pain, neck mass	Right and left posterior mediastinum	Submitted slide of cervical lymph node	6-48	Cervical lymph node excision	9-48	Skull and pelvic metastases
Case of Paraganglioma									
24. J. H.	M	52	Fatigue, cough (blood pressure normal)	Left upper posterior mediastinum	Encapsulated, soft, red-tan mass 5.5 by 5.5 by 3.5 cm.; cut surface red-gray, soft and homogenous	10-49	Total excision	12-49	Asymptomatic, negative x-ray

tinal xanthomas[10] appear to represent neurogenic tumors (encapsulated neurilemoma) that had a prominent number of lipophages. The number of lipoid-filled histiocytes was marked in several of the tumors in this series.

CLINICAL MATERIAL

The 24 patients on which this report is based include 14 women and 10 men, varying in age from 5 to 57 years (Table I). In 10 instances the lesion was incidentally found during Army separation examinations, tuberculosis surveys, or examinations for other illness. Among the remainder, cough and dyspnea were the most common initial symptoms. Even in the large tumors (Fig. 1, *A* and *B*), pain was not a prominent feature. No instance of "dumbbell tumor" with spinal cord compression was encountered.



A. *B.*

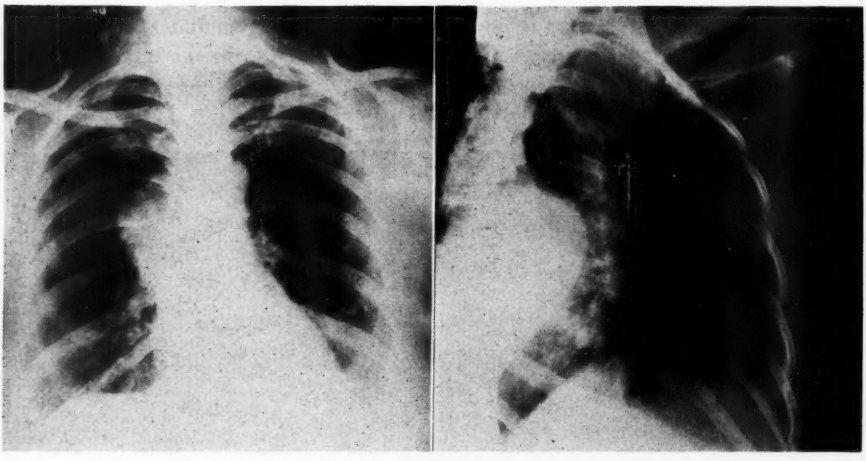
Fig. 1, *A* and *B* (Case 20, M. E.).—Ganglioneuroma showing displacement of the heart but no scoliosis.

The paravertebral gutter is the commonest site for intrathoracic tumors of nerve origin. A diffuse neurilemoma (major component of a teratoma) was encountered in the anterior mediastinum (Case 1) and 2 encapsulated neurilemomas on the inner lateral chest wall. By long usage the paravertebral tumors are considered mediastinal. In this series, 3 ganglioneuromas extended across the anterior surface of the vertebral bodies in the true mediastinum.

The diagnosis of a neurogenic tumor is relatively certain by radiographic examination when a spherical or lobulated shadow is seen in the paravertebral gutter. The finding of stigmas of diffuse neurilemomatosis (von Recklinghausen's disease) enhances the diagnosis of a neurogenic tumor. Bone changes consist of widening of intercostal spaces and erosion of adjacent ribs and

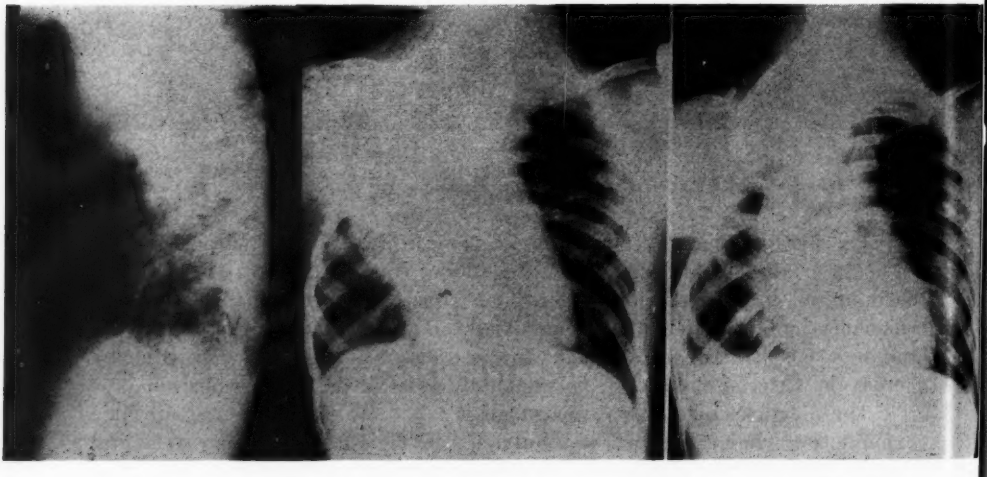
vertebrae (Fig. 2), due to pressure. There may be enlargement of the vertebral foramina even in the absence of spinal cord signs. Other lesions to be considered are: chondrosarcoma of rib, lipoma,[11] meningocele,[12] aneurysm of descending thoracic aorta, and esophageal achalasia.

Twenty-two patients were operated upon without a fatality. All the encapsulated neurilemomas and the solitary paraganglioma were totally excised, but only 3 of the 8 ganglioneuromas were so treated. Three gang-



A. *B.*

Fig. 2, *A* and *B* (Case 17, R. H.).—Ganglioneuroma showing separation of ribs and erosion of vertebral bodies.



A. *B.* *C.*

Fig. 3.—*A* and *B* show an extensive lobulated malignant neurilemoma. *C* shows postoperative appearance with an apparent metastasis in the right lower lung field.

lioneuromas were subjected to partial excision due to extension of the growth between ribs, along vertebral surfaces, and in one case into the lower neck. The malignant neurilemoma could not be totally removed from the right chest due to encirclement of the trachea (Fig. 3), and only tissue for biopsy was taken from 2 ganglioneuromas, the diffuse neurilemoma, and from a metastatic focus in the case of the sympathicoblastoma.

Fig. 4 (Case 21, J. M.).—A fusiform ganglioneuroma displacing the aorta and esophagus to the right.

Surgical technique of extirpation must usually involve a transpleural approach for adequate visualization, preferably through a posterolateral rib bed at the level of the tumor. In children an intercostal incision will avoid scoliosis. The major technical difficulty concerns hemostasis because the branches of the intercostal vessels adhere to the tumor surface, and are present on the side of the growth opposite the operator. Preliminary incision of the parietal pleura completely around the growth and elevation off adjacent ribs will usually allow enough mobility so that the intercostal vessels are stretched and can be visualized crossing vertebral bodies or, on the left, immediately adjacent to the aorta permitting ligation and division under direct visualization. In certain tumors, total removal is impractical due to extension into the neck, across the midline (Fig. 4), or because of sheer bulk; then partial resection is resorted to. No radiographic or clinical evidence of further growth has followed biopsy in 2 instances and partial excision in 3 other ganglioneuromas, the longest now followed over four years (Case 15). Less wide total removal naturally results in failure where one is dealing with a malignant neurogenic tumor. Irradiation does not offer permanent arrest of this tumor.

PATHOLOGY

The tumors included in this study have been classified according to the following scheme, and will be discussed in this order. The number of examples of each is indicated at the right.

I. Tumors of nerve sheath origin	
A. Diffuse neurilemoma*†	1‡
B. Encapsulated neurilemoma	12
C. Malignant neurilemoma	1
II. Tumors of sympathetic origin	
A. Ganglioneuroma	8
B. Sympathicoblastoma	1
III. Tumors of paraganglionic tissue	
A. Paraganglioma	1

For a more detailed classification of tumors of peripheral nerves, the reader is referred to a fascicle on the subject by Dr. A. P. Stout, prepared under the auspices of the Armed Forces Institute of Pathology.

TUMORS OF SHEATH ORIGIN

One of the many problems in dealing with tumors of peripheral nerves, and, in particular, the tumors of sheath origin, is their classification. In the laboratories of Memorial Hospital the view has long been held that these tumors are derived from Schwann cells.[14, 15] Nageotte[16] experimentally demonstrated the schwannian origin of the encapsulated neurilemoma in rabbits. Masson and Simard[17] and Masson,[18] stimulated by the work of Nageotte, compared experimentally produced encapsulated neurilemomas with spontaneous encapsulated neurilemomas of man, and were convinced that these tumors were constituted almost exclusively of Schwann cells. This belief has been supported and expanded through tissue culture studies to include the diffuse neurilemoma and malignant neurilemoma.[19, 20, 21, 22]

Stout[23] has suggested that the term "diffuse neurilemoma" be employed for the tumor commonly called neurofibroma and for the discrete lesion, the expression "encapsulated neurilemoma." This proposal appears useful to us, because it indicates, on the one hand, histogenetic relationship and, on the other, an anatomic difference. The histologic distinction is certainly not an absolute one, since minor attributes of one lesion may be present in the other, for example, small encapsulated neurilemomas in what is otherwise a diffuse neurilemoma (neurofibroma or plexiform neuroma).

The tumor here designated as diffuse neurilemoma has been most commonly referred to in the past as neurofibroma. Other terms employed include myxoid neurinoma, neurilemoblastoma, neurinoma, plexiform neurinoma, cirsoid neuroma, multiple neurofibromatosis, and von Recklinghausen's disease.

These tumors are seen in most classical form as a manifestation of von Recklinghausen's disease. Characteristically, the lesion may produce beading

*Vieta and Pack[13] have employed the term "Plexiform neurilemoma" to indicate the lesion herein described as diffuse neurilemoma.

†The designation "schwannoma" may be used synonymously with neurilemoma.

‡This tumor was a teratoma growing principally as a diffuse neurilemoma.

or diffuse tortuous enlargement of nerve trunks and multiple or solitary nonencapsulated cutaneous tumors.

Histologically, the tumors are composed of Schwann cells, neurites, and fibrous tissue. The neurites are likely to be scanty.

Metaplasia (cartilage and bone) and more differentiated elements (adult ganglion cells, replicas of Wagner-Meissner tactile corpuscle) may occasionally be noted. The microscopic picture (Fig. 5) may be differentiated from that of the encapsulated neurilemoma by the haphazard arrangement of the Schwann cells, prominent reticulation of the intercellular material, absence of palisading, and other features to be mentioned.

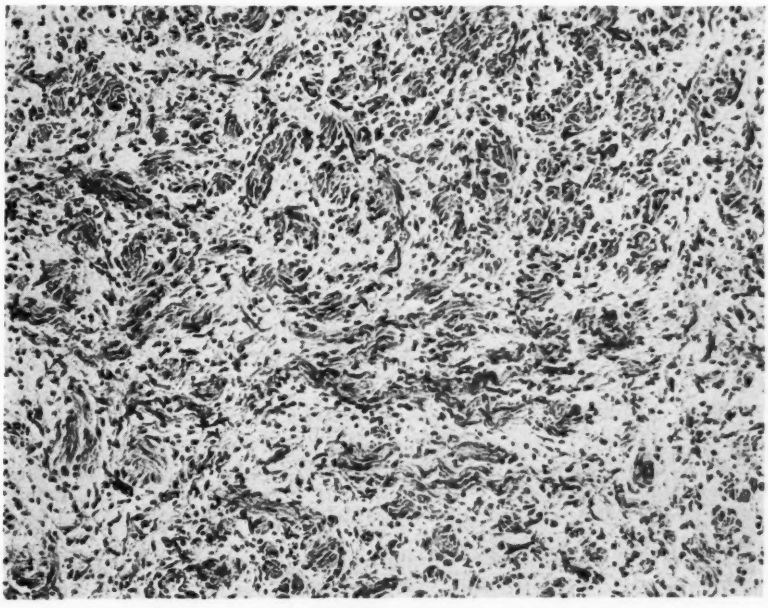


Fig. 5.—Diffuse neurilemoma.

It is important to distinguish the diffuse from the encapsulated neurilemoma because about 13 per cent[24] of diffuse neurilemomas become malignant, whereas encapsulated neurilemomas rarely, if ever, do become malignant. Vieta and Pack[13] are of the opinion that the incidence of malignant change in diffuse neurilemoma is less than 13 per cent. Recently Saxén[25] reported one case which he believed probably represented malignant change in an encapsulated neurilemoma. An occasional case of recurrent or locally infiltrating encapsulated neurilemoma has been reported, as cited by Ehrlich and Martin.[26]

The encapsulated neurilemoma (synonyms: schwannoma, specific nerve sheath tumor, neurinoma, perineural fibroblastoma, peripheral glioma, schwannoglioma, palisaded neurinoma, acoustic neuroma) usually presents as a solitary, encapsulated tumor attached to a nerve from which it may usually be removed without serious damage. Occasionally, however, a tumor is encoun-

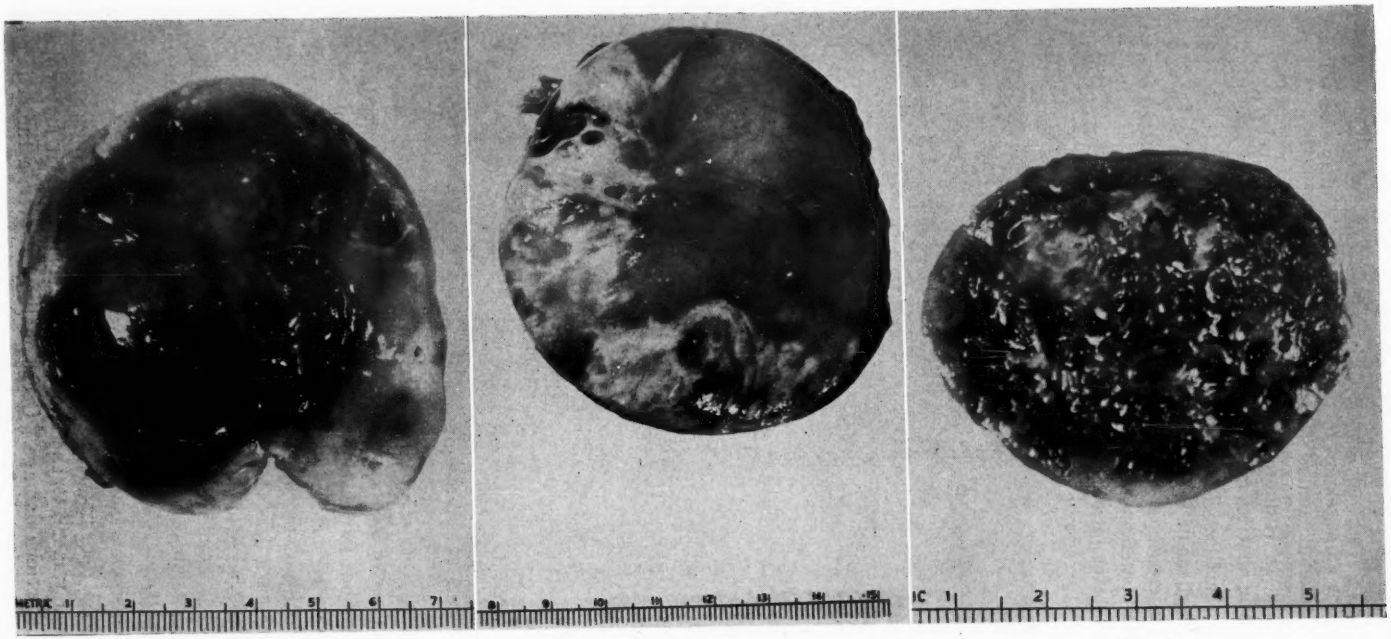


Fig. 6.

Fig. 7.

Fig. 8.

Fig. 6.—Encapsulated neurilemoma with areas of hemorrhage and necrosis.
Fig. 7.—Encapsulated neurilemoma. Light areas indicate zones of lipophages.
Fig. 8.—Encapsulated neurilemoma containing numerous vascular spaces simulating hemangioma.

tered within a nerve, making resection of the nerve compulsory for adequate removal. This lesion is usually not associated with von Recklinghausen's disease, although about 18 per cent[27] occur in frank or suspected cases of this disease. Ehrlich and Martin[26] found none of the stigmas of von Recklinghausen's disease in 12 cases of encapsulated neurilemoma of the head and neck. Material reviewed at Memorial Hospital by Vieta[28] indicated that the incidence of encapsulated neurilemoma in von Recklinghausen's disease is lower than 18 per cent. These tumors may reach huge proportions in the mediastinum and abdomen and undergo hemorrhage and cystic changes (Fig. 6). They may be yellow due to sudanophilic material (Fig. 7) or hemangiomatous because of numerous patulent vessels (Fig. 8). Microscopically, they are composed of Schwann cells, which may form Verocay nuclear rows (Antoni A) (Fig. 9), reticulated tissue (Antoni B) (Fig. 10), or a combination of the two. This last is the most frequent. A few neurites may be found near the former attachment to the nerve. Fibrous tissue varies in amount from area to area and tumor to tumor. Small round cells, plasma cells, and mast cells are seen in varying numbers. The blood supply may be so prominent that it suggests hemangioma (Fig. 11). The individual vessels show collagen collars, thrombosis, and subintimal lipophages. Extravasation of blood apparently occurs, since hemosiderin is occasionally present (Table II).

TABLE II. CERTAIN FEATURES OF ENCAPSULATED NEURILEMOMAS

	LIPOPHAGES	PLEOMORPHISM	HEMOSIDERIN	VESSEL COLLARS
12 cases	8	8	4	10

Large numbers of lipophages (Fig. 12) may be found producing a pseudoxanthomatous appearance. The source of this fatty material is doubtful. It may be the result of hemorrhage or possibly be phagocytized myelin from degenerated myelinated fibers. Similar areas of lipophages are seen in these tumors in other locations.

Besides Antoni A and B and mixed encapsulated neurilemomas, the mediastinal tumors of this series included examples with an additional pattern. Several of the tumors were composed of closely packed, interlacing, and haphazardly arranged cells containing large hyperchromatic nuclei (Fig. 13). These features were so prominent that two of the lesions were classed as having equivocal changes of a type that might lead one to anticipate the possibilities of a recurrence and another was diagnosed as malignant neurilemoma (neurosarcoma). By virtue of adequate follow-up study these tumors are now known to be merely a pleomorphic variety of the encapsulated neurilemoma. It is this lesion that is most likely to be confused with malignant neurilemoma. Lesions from other locations may show a similar pleomorphic pattern and yield similar confusion.

Malignant neurilemoma (synonyms: neurogenic or neurogenous sarcoma, fibrosarcoma of nerve sheath, malignant neurinoma, malignant schwannoma) a tumor of Schwann cells, may arise apparently de novo or in diffuse neurilemoma (Fig. 14, *A* and *B*). It is generally considered not to occur in the encapsulated neurilemoma.[27, 29] In a large series of neurogenic sarcomas (malignant

Fig. 9.

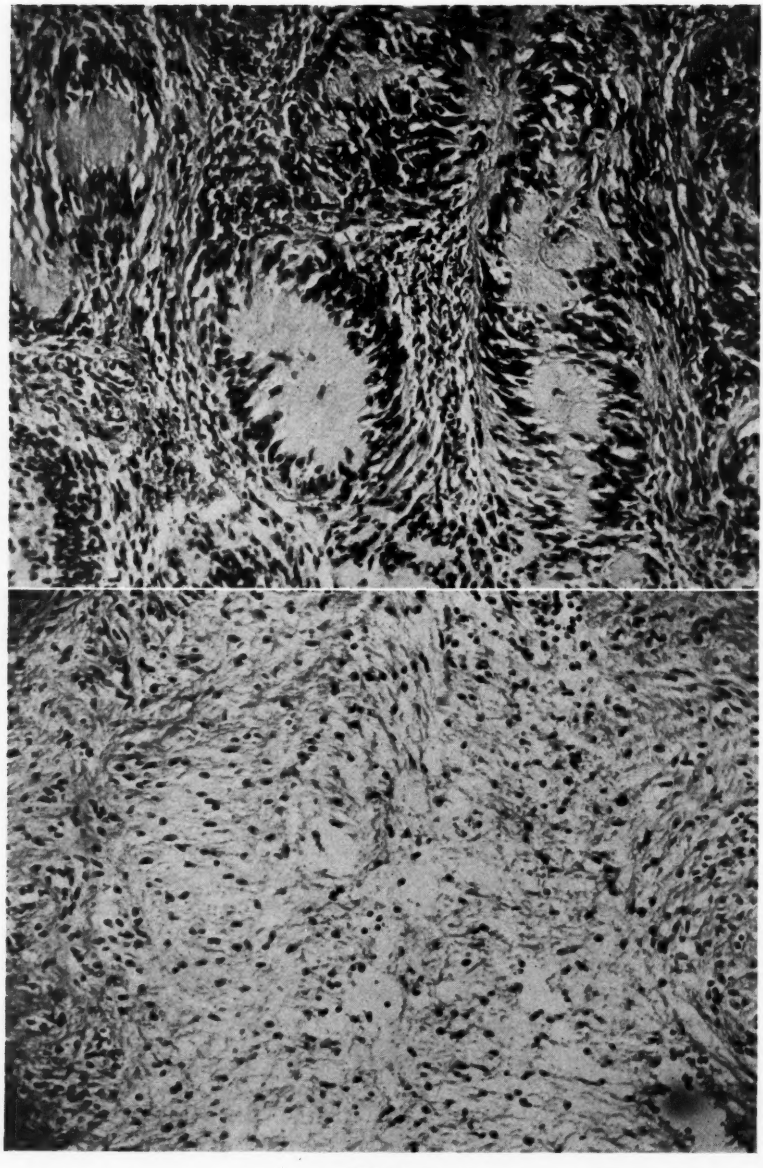


Fig. 10.

Fig. 9.—Encapsulated neurilemoma with palisading of nuclei (Antoni A).
Fig. 10.—Encapsulated neurilemoma with reticulated (Antoni B) areas.

Fig. 11.

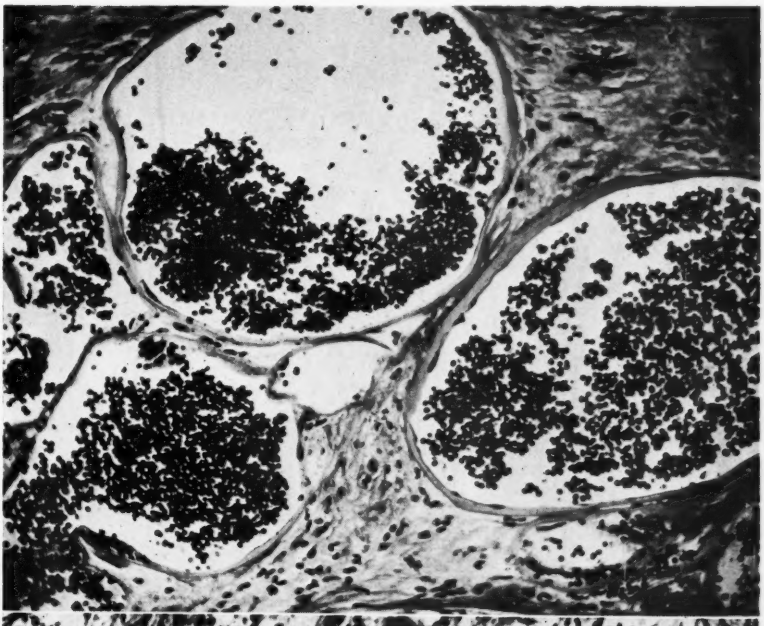


Fig. 12.

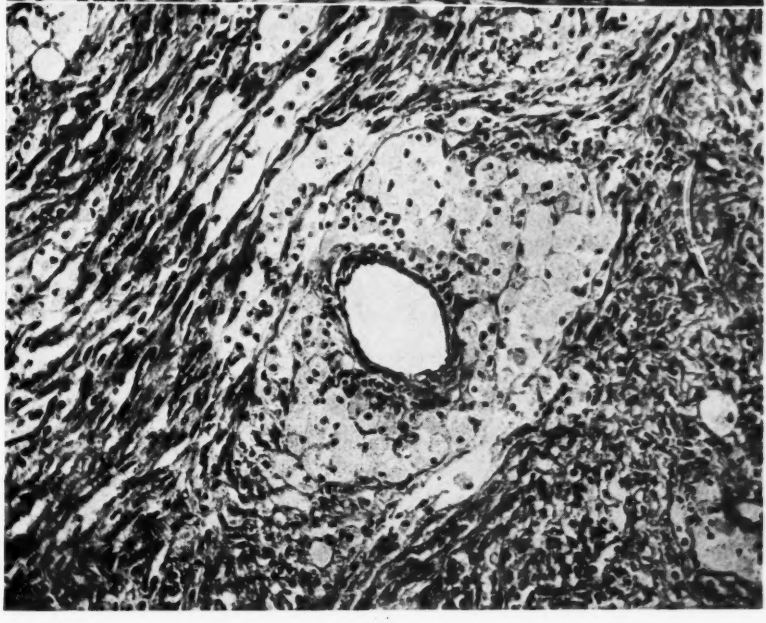
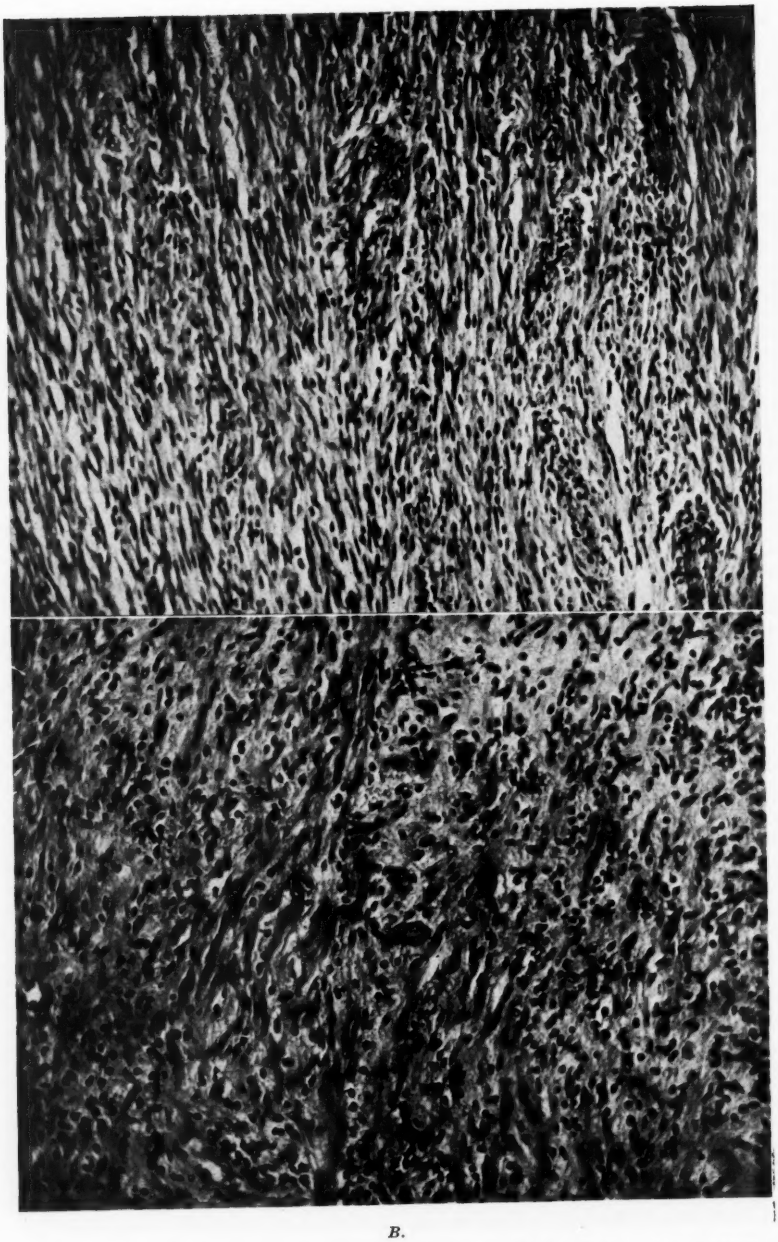


Fig. 11.—Encapsulated neurilemoma demonstrating thick vessel walls.
Fig. 12.—Encapsulated neurilemoma with perivascular lipophages.

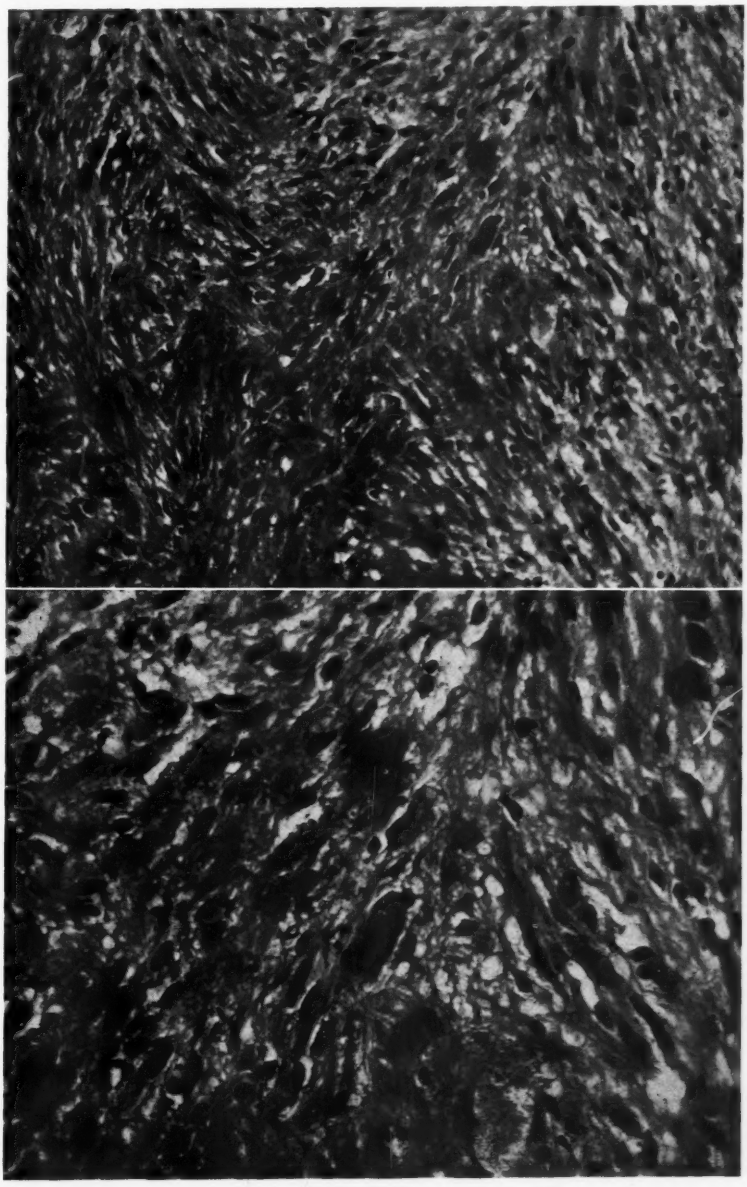
A.



B.

Fig. 13. *A* and *B*.—Encapsulated neurilemomas showing cellular pleomorphism.

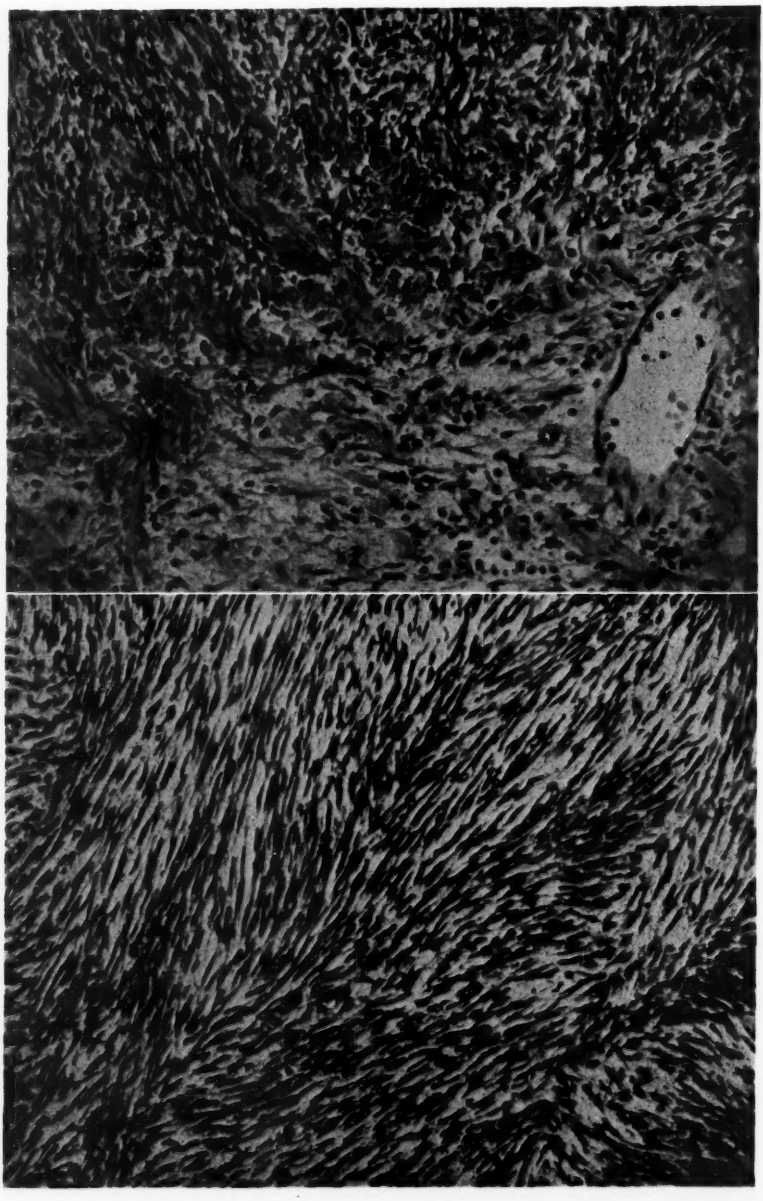
C.



D.

Fig. 13, *C* and *D*.—Encapsulated neurilemomas showing cellular pleomorphism.

A.

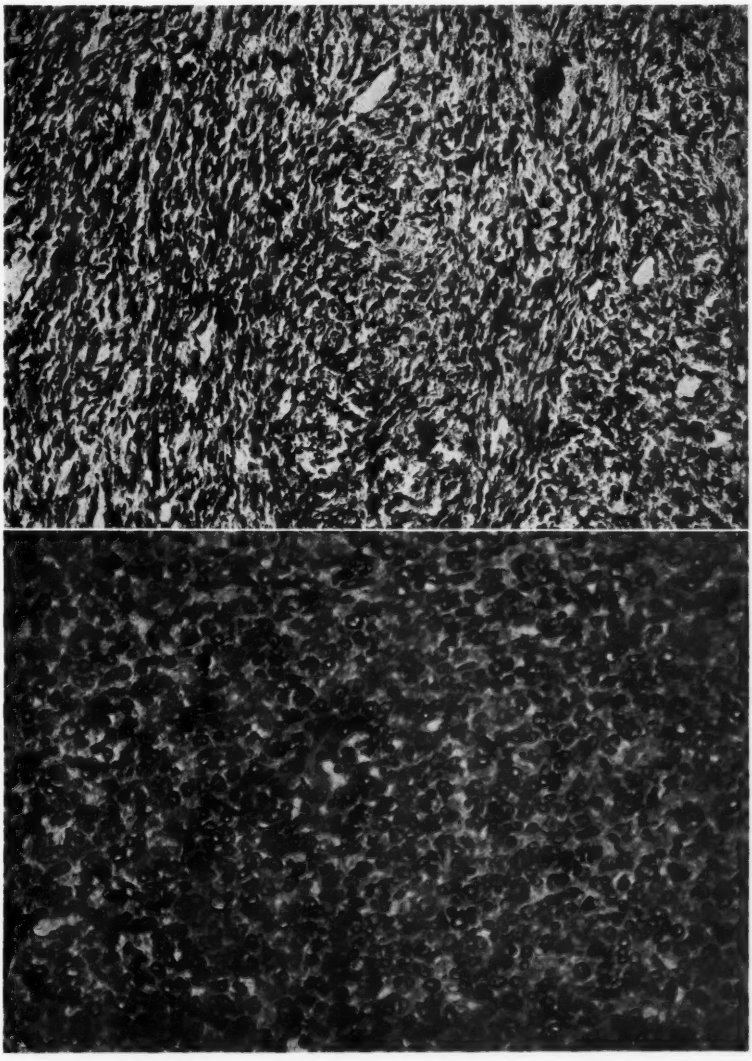


B.

Fig. 14.—*A*, Junction of diffuse neurilemoma and malignant neurilemoma. *B*, Malignant neurilemoma showing interlacing fascicles of spindle cells.

neurilemoma) Stewart and Copeland[15] found that about one-quarter were in patients having manifestations of von Recklinghausen's disease. Vieta and Pack[13] found stigmas of von Recklinghausen's disease in 11 of 31 cases of malignant neurilemoma.

A.



B.

Fig. 15.—*A*, Malignant neurilemoma, low grade. *B*, Malignant neurilemoma with extreme anaplasia. From same patient as *A*. Another portion of the same tumor. Case of generalized von Recklinghausen's disease.

Demonstration of nerve origin may at times be quite difficult. However, familiarity with the growth pattern may enable one to assume a nerve origin with reasonable assurance in many instances.

Fig. 16.

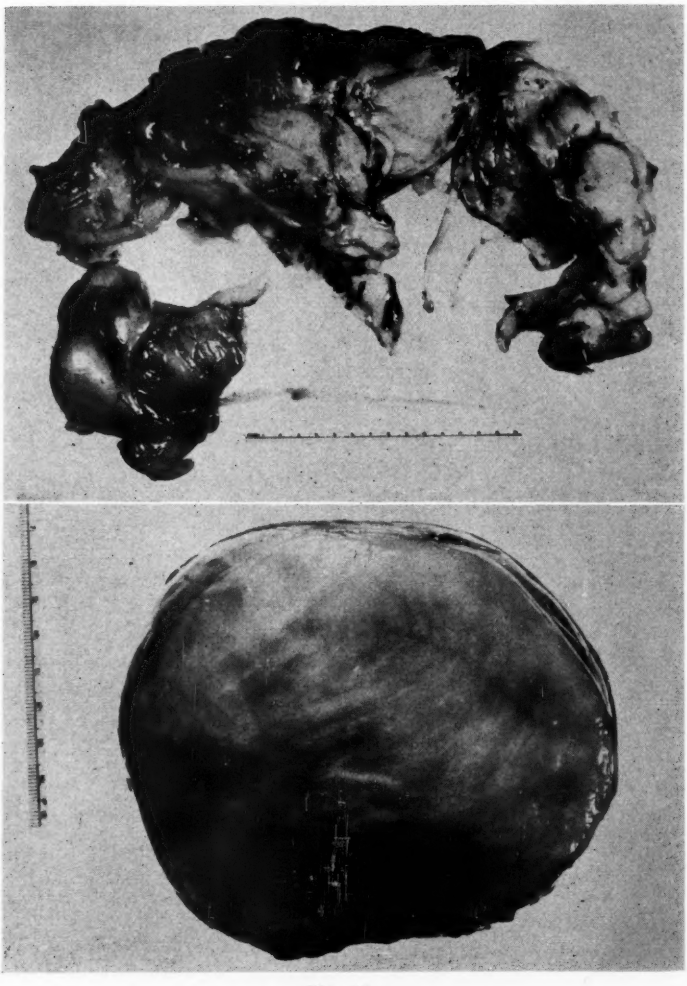


Fig. 17.

Figs. 16 and 17.—Ganglioneuroma.

Grossly, the lesions are encapsulated or circumscribed. Microscopically, the picture varies from the relatively acellular fibrous tumor to one with prominent or even extreme cellularity (Fig. 15, *A* and *B*). Where cellularity is extreme many pathologists are reluctant to assert the neurogenous nature of the

tumor, especially when history and gross anatomic data are lacking. Masson's trichrome and reticulum stains are regarded by many as helpful in arriving at diagnosis.

TUMORS OF SYMPATHETIC GANGLIA

Ganglioneuromas may occur in any location containing ganglion cells, but most frequently arise from the thoracic and abdominal sympathetic chains. Stout[30] recently reported an analysis of 243 cases of which 62 were mediastinal. He had separated the tumors into differentiated and undifferentiated types depending upon the presence of immature nerve cells.

Grossly, the tumors may be quite large, soft to firm, yellow to gray, and encapsulated (Figs. 16 and 17). Occasionally, there is tumor both in the thorax and in the spinal canal, the so-called hourglass tumor. Microscopically, the differentiated ganglioneuroma is composed of mature ganglion cells set in a stroma of myelinated and nonmyelinated sheathed nerve fibers (Fig. 18, *A* and *B*). The ganglion cells may be numerous or sparse and show various degenerative changes. Foci of small round cells may be scattered about; at times these have been considered neuroblasts (Fig. 19). This conclusion, however, does not appear tenable from present observations, since no differentiation of these cells was noted, and, morphologically, they resemble lymphoid cells. Also, foci of similar cells were seen in the neurilemomas. Murray and Stout[31] have made tissue culture studies of a ganglioneuroma.

The undifferentiated type may contain either partly differentiated neuroblasts, in which case the incidence of metastasis is 18.2 per cent, or areas of frank neuroblastoma yielding metastasis in 65 per cent.[30]

Sympathicoblastoma[32, 33, 34, 35, 36] (synonyms: neurocytoma, neuroblastoma, ganglioma embryonale sympathicum, sympathicogonioma) although frequently arising in the adrenal medulla, occasionally arises from the sympathetic system in other locations.

Grossly, the tumor is soft, with or without capsule, and varies in consistency and color depending upon degenerative changes.

Microscopically (Fig. 20), the picture is varied depending upon the degree and type of differentiation of sympathicogonia. The lesions have been divided according to the most prominent cell type, presence of axons, and the formation of rosettes. This is of questionable value, since the prognosis is poor in all, and the variety of names merely adds to the state of confusion. We, therefore, prefer the term "sympathicoblastoma" for the various patterns of this cancer.

TUMORS OF PARAGANGLION CELLS

Boyd[37] stated that the term "paraganglia"[38] indicated a structure, the essential cells of which are derived in their entirety from the nervous system and, to have significance, these essential cells must arise from primitive cells identical with, and of the same genetic origin as, forerunners of true ganglion cells. The cells destined to become paraganglion cells migrate to the dorsal surface of the sympathetic ganglia to form small, rounded masses in depressions of these ganglia, therefore, the term "paraganglion." These cells reduce chromates and this is known as the pheochrome reaction. This feature, how-

ever, is nonspecific.[39, 40, 41] "While by definition it is possible for a paraganglion to arise from any part of the nervous system, our knowledge of concrete examples of paraganglia is confined to those which develop from the

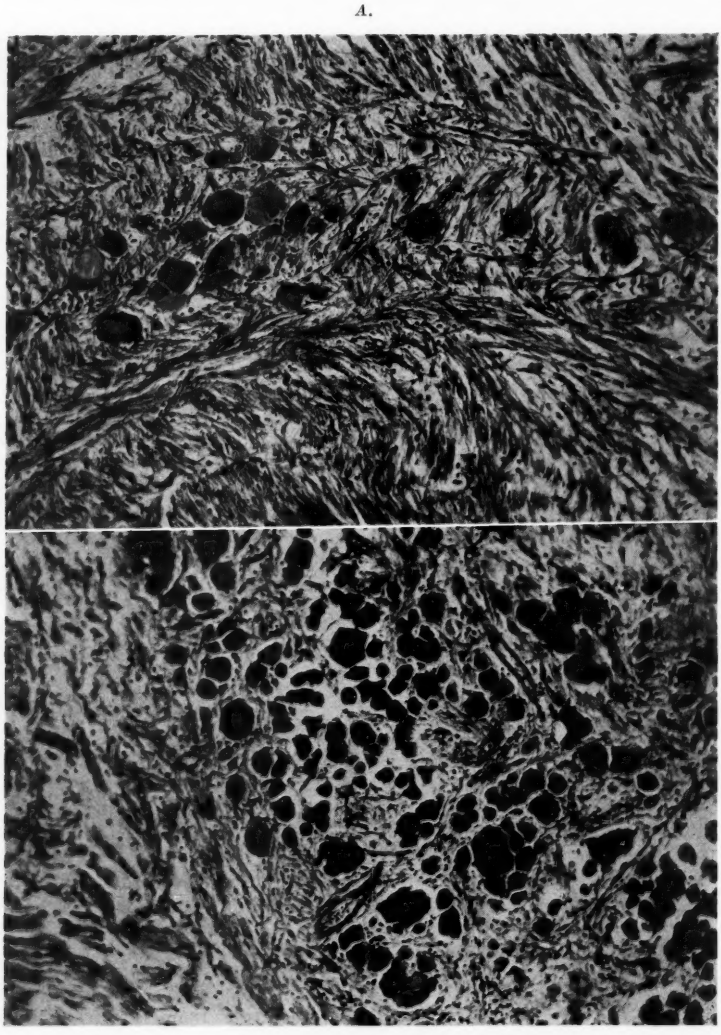


Fig. 18.—*A*, Ganglioneuroma showing general pattern. *B*, Ganglioneuroma showing variability of ganglion cells.

embryonic sympathetic nervous system—the adrenal medulla, the aortic bodies of Zuckerkandl and the chromaffin tissue found in the sympathetic ganglia."[37] The term "paraganglia" may be used in a wider sense, including paraganglia that originate from other portions of the peripheral nervous system, such as

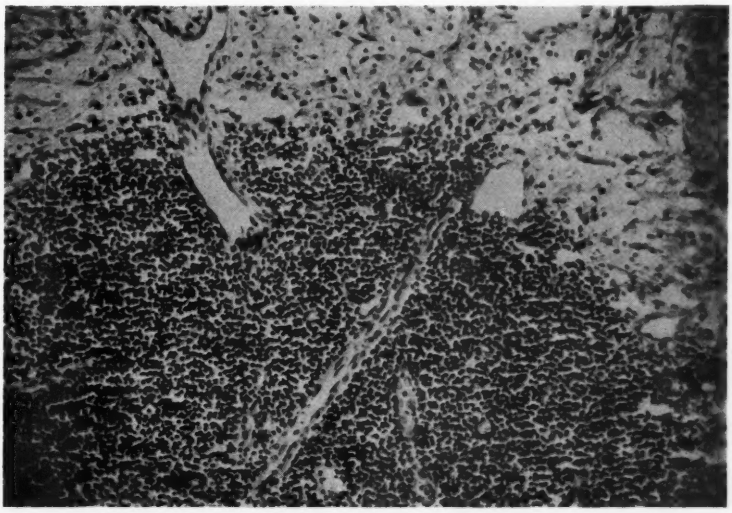


Fig. 19.—Focus of lymphoid cells found in neurilemoma and ganglioneuroma.

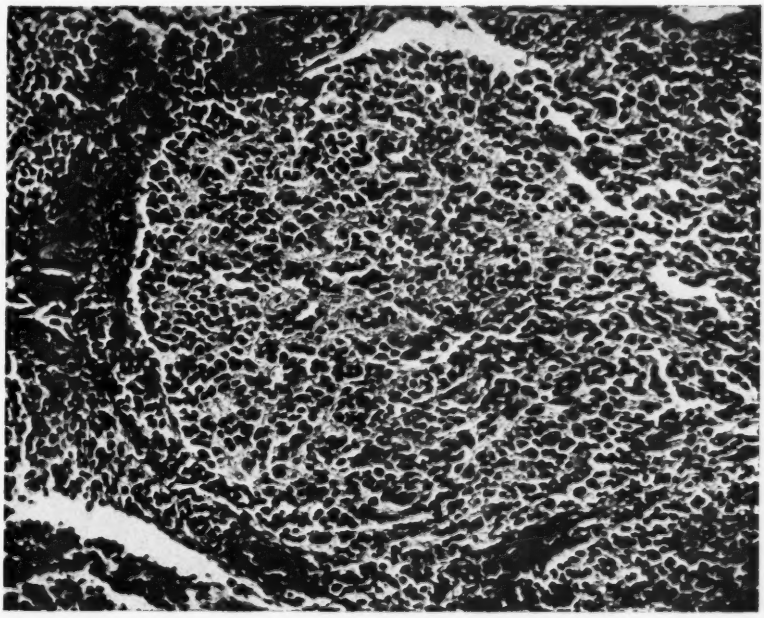
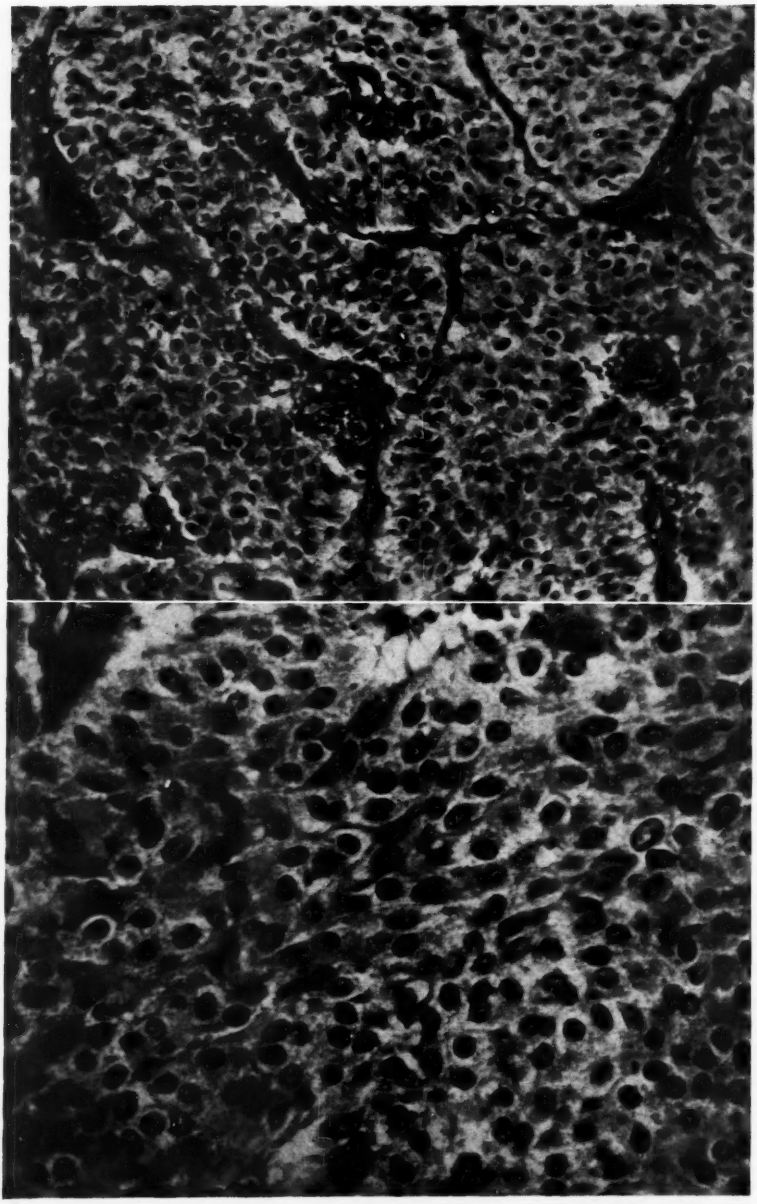


Fig. 20.—Sympathicoblastoma showing general pattern.

A.



B.

Fig. 21.—*A*, Paraganglioma showing general pattern. *B*, Paraganglioma showing uniform nuclei and indefinite cell walls.

the cranial nerves. We agree with Brines and Jennings[41] that the designation "paraganglioma" is preferable to pheochromocytoma or chromaffinoma owing to nonspecificity of the reduction of chromates, the absence of this reaction in many tumors, and the known histogenetic origin from paraganglion cells. These authors accept two tumors of previously reported intrathoracic paraganglioma. Recently, Maier[42] added another case. One other unreported case of the posterior mediastinum has been examined by one of us (J.T.G.).[43]

Grossly, the tumors may be large, encapsulated, reddish brown, soft, and show areas of necrosis.

The histologic picture varies from one resembling the adrenal medulla to one that is so atypical that diagnosis is a problem.

The cells may be arranged in cords or sheets and separated by a fine connective tissue (Fig. 21, *A* and *B*). The vascular component is usually abundant. The pheochrome reaction may or may not be positive and they may or may not be hormonally active. No malignant intrathoracic paragangliomas have been reported. However, McGavock and associates[44] accepted 8 cases of malignant paraganglioma from other anatomic locations. Cross and Pace[45] reported an additional case.

It should be mentioned that carotid-body tumors may be morphologically similar to paragangliomas; however, Boyd[37] is convinced that the carotid body and also the aortic-arch bodies should not be included as paraganglia.

Aortic-arch body tumors have been reported in dogs[46] and should be anticipated in man. Lattes[47] is reporting one aortic-arch body tumor in a paper on nonchromaffin paragangliomas to be published in *Cancer*. Several cases (dog) have been examined by one of us (J.T.G.) through the courtesy of Dr. R. Mulligan. Lattes and Waltner[48] have designated carotid-body tumors, glomus-jugulare tumors, and tumors of morphologically related tissues as nonchromaffin paragangliomas. This term could be applied to the tumor herein reported; however, as mentioned, the pheochrome reaction is nonspecific and not all true paragangliomas give the reaction. The identity of the morphologically related tissues as being true paraganglia is not established, in particular, the carotid and aortic-arch bodies as shown by Boyd.

Further work is essential for the clarification of histogenesis and function of these morphologically related tissues.

DISCUSSION AND CONCLUSIONS

The intrathoracic nerve-sheath tumors are predominantly encapsulated neurilemomas, of which there were 12 cases. They are benign, rarely, if ever, become malignant, and offer a good prognosis. One diffuse neurilemoma was encountered as the principal component of a teratoma. This patient died as the result of continued growth of the teratoma. One instance of malignant neurilemoma was found associated with von Recklinghausen's disease (diffuse neurilemoma) and this eventually resulted in the death of the patient.

Of the nerve-cell tumors there were 8 ganglioneuromas, all of which were differentiated. The prognosis of this lesion is good following removal. More adequate follow-up of this lesion is necessary in order to ascertain the prognosis

in instances in which all of the tumor tissue has not been removed. A malignant course is not to be expected if the tumor is of the differentiated variety, which is the usual type found in the mediastinum.

There was one case of sympathicoblastoma thought to be primary in the mediastinum but not proved by autopsy. One paraganglioma was encountered that was hormonally inactive and pheochrome negative. This was a benign tumor and the prognosis is good. The possibility of this lesion being an aortic-arch body tumor was considered; however, it appeared too intimately related to the sympathetic chain to be other than a paraganglioma.

In this series of 24 neurogenic tumors 2 (8.33 per cent) were malignant.

SUMMARY

Twenty-four primary intrathoracic neurogenic tumors are reported with special emphasis on the pathologic features and classification. Two of the 24 cases were malignant. A new case of intrathoracic paraganglioma is included.

REFERENCES

1. Kent, E. M., Blades, B., Valle, A. B., and Graham, E. A.: Intrathoracic Neurogenic Tumors, J. THORACIC SURG. **13**: 116-161, 1944.
2. Blades, B.: Mediastinal Tumors, Ann. Surg. **123**: 749-765, 1946.
3. Curreri, A. R., and Gale, J. W.: Mediastinal Tumors, Arch. Surg. **58**: 797-818, 1949.
4. Brewer, L. A., and Dolley, F. S.: Tumors of the Mediastinum, Am. Rev. Tuberc. **60**: 419-438, 1949.
5. Harrington, S.: Intrathoracic Extrapulmonary Tumors: Diagnosis and Surgical Treatment, Postgrad. Med. **6**: 6-21, 1949.
6. Arbuckle, R. K.: Solitary Tumors of the Chest: The Differential Diagnosis in Fifty Proved Cases, Am. J. Roentgenol. **62**: 52-64, 1949.
7. Bartlett, J. P., and Adams, W. E.: Solitary Primary Neurogenic Tumor of the Lung. Report of a Case, J. THORACIC SURG. **14**: 251-260, 1945.
8. Touroff, A. S. W., and Sapin, S. O.: Solitary Intrathoracic Neurofibroma, Surgery **26**: 787-798, 1949.
9. Klassen, K. P., Patton R., and Bemen, F. M.: Neurofibroma of the Diaphragm, J. THORACIC SURG. **14**: 407-413, 1945.
10. Phillips, E. W.: Intrathoracic Xanthomatous New Growths. Report of Two Cases, J. THORACIC SURG. **7**: 74-95, 1937.
11. Watson, W. L., and Urban, J. A.: Mediastinal Lipoma, J. THORACIC SURG. **13**: 16-29, 1944.
12. Mendelsohn, H. J., and Kay, E. B.: Intrathoracic Meningocele, J. THORACIC SURG. **18**: 124-128, 1949.
13. Vieta, J. O., and Pack, G. T.: The Malignant Tumors of Peripheral Nerves. In press.
14. Ewing, James: Neoplastic Diseases, ed. 4, Philadelphia, 1940, W. B. Saunders Company, pp. 161-172.
15. Stewart, F. W., and Copeland, M. M.: Neurogenic Sarcoma, Am. J. Cancer **15**: 1235-1320, 1931.
16. Nageotte, J.: Sheaths of the Peripheral Nerves. Nerve Degeneration and Regeneration. Cytology and Cellular Pathology of the Nervous System. Penfield, Wilder, Vol. 1, New York, 1932, Paul B. Hoeber, Inc., pp. 189-239.
17. Masson, P., and Simard, C.: Spontaneous and Experimental Schwannomas, Am. J. Path. **6**: 618, 1930.
18. Masson, P.: Experimental and Spontaneous Schwannomas (Peripheral Gliomas), Am. J. Path. **8**: 367-416, 1932.
19. Geschickter, C. F.: Tumors of the Peripheral Nerves, Am. J. Cancer **25**: 377-410, 1935.
20. Murray, M. R., Stout, A. P., and Bradley, C. F.: Schwann Cell Versus Fibroblast as the Origin of the Specific Nerve Sheath Tumor, Am. J. Path. **16**: 41-60, 1940.
21. Murray, M. R., and Stout, A. P.: Characteristics of Human Schwann Cells in Vitro, Anat. Rec. **84**: 275-294, 1942.
22. Murray, M. R., and Stout, A. P.: Demonstration of the Formation of Reticulin by Schwannian Cells in Vitro, Am. J. Path. **18**: 585-589, 1942.
23. Stout, A. P.: Neurofibroma and Neurilemoma, Clin. Proc. **5**: 1-12, 1946.
24. Hosoi, K.: Multiple Neurofibromatosis (von Recklinghausen's Disease), Arch. Surg. **22**: 258-281, 1931.

25. Saxén, E.: Tumors of the Sheaths of the Peripheral Nerves, Acta path. et microbiol. Scandinav. (suppl.) **79**: 1948.
26. Ehrlich, H. E., and Martin, Hayes: Schwannomas (Neurilemomas) in the Head and Neck, Surg., Gynec. & Obst. **76**: 577-583, 1943.
27. Stout, A. P.: The Peripheral Manifestations of the Specific Nerve Sheath Tumor (Neurilemoma), Am. J. Cancer **24**: 751-796, 1935.
28. Vieta, J. O.: Unpublished data.
29. Stewart, F. W.: Personal communication.
30. Stout, A. P.: Ganglioneuroma of the Sympathetic Nervous System, Surg., Gynec. & Obst. **84**: 101-110, 1947.
31. Murray, M. R., and Stout, A. P.: A Sympathetic Gangioneuroma Cultivated in Vitro, Cancer **1**: 242-247, 1948.
32. Lewis, D., and Geschickter, C. F.: Tumors of the Sympathetic Nervous System: Neuroblastoma, Paraganglioma, Ganglioneuroma, Arch. Surg. **28**: 16-58, 1934.
33. Wahl, H. R., and Robinson, D. W.: Neuroblastoma of Mediastinum With Pheochromoblastomatous Elements, Arch. Path. **35**: 571-578, 1943.
34. Wright, J. H.: Neurocytoma or Neuroblastoma—A Kind of Tumor Not Generally Recognized, J. Exper. Med. **12**: 556-561, 1910.
35. Wahl, H. R.: Neuroblastoma: With a Study of a Case Illustrating These Three Types That Arise From the Sympathetic System, J. Med. Research **30**: 205-260, 1914.
36. Hollingsworth, R. K.: Intrathoracic Tumors of the Sympathetic Nervous System, Surg., Gynec. & Obst. **82**: 682-693, 1946.
37. Boyd, J. D.: The Development of the Human Carotid Body. Contributions Embryol. **26**: 1-31, 1937.
38. Hollinshead, W. H.: Chromaffin Tissue and Paraganglin. Quart. Rev. Biol. **15**: 156-171, 1940.
39. Kingsbury, B. F.: The Term "Chromaffin System" and the Nature of the Chromaffin Reaction, Anat. Rec. **5**: 11-16, 1911.
40. Le Compte, P. M.: Tumors of the Carotid Body, Am. J. Path. **24**: 305-321, 1948.
41. Brines, O. A., and Jennings, E. R.: Paragangliomas: Review of Subject and Report of Five Original Cases, Am. J. Path. **24**: 1167-1197, 1948.
42. Maier, H. C.: Intrathoracic Theochromocytoma With Hypertension, Ann. Surg. **130**: 1059-1065, 1949.
43. Olson, R., Jacobson, G., Finefield, A., and Brewer, L.: To be reported.
44. McGavock, T. H., Benjamin, J. W., Speer, F. D., and Klotz, S.: Malignant Pheochromocytoma of the Adrenal Medulla (Paraganglioma). Report of Case Simulating Carcinoma of Adrenal Cortex With Secondary Adrenal Insufficiency, J. Clin. Endocrinol. **2**: 332-338, 1942.
45. Cross, G. O., and Pace, J. W.: Malignant Pheochromocytoma With Paroxysmal Hypertension and Metastasis to the Cervical Spine, J. A. M. A. **142**: 1068-1070, 1950.
46. Bloom, F.: Structure and Histogenesis of Tumors of the Aortic Bodies In Dogs, Arch. Path. **36**: 1-12, 1943.
47. Lattes, R.: Nonchromaffin Paraganglioma of Ganglion Nodosum, Carotid-body and Aortic-arch Bodies, Cancer **3**: 667-694, 1950.
48. Lattes, R., and Waltner, J. G.: Nonchromaffin Paraganglioma of the Middle Ear (Carotid Body-Like Tumor; Glomus Jugulare Tumor) Cancer **2**: 447-468, 1949.

DISCUSSION

DR. HERBERT C. MAIER, New York.—There are a few points that I should like to emphasize. We have had 3 cases of benign neurogenic tumor in the apex of the thorax that have extended into the neck and encircled the subclavian artery. In such cases the approach through the thorax may be relatively unsatisfactory, because one is unable to remove the cervical portion. Therefore, in the last of these 3 patients I employed an incision similar to a posterior thoracoplasty incision, except that I extended it further upward, and was able thus to remove that part of the tumor which extended into the posterior cervical region as well as the main mass through an extrapleural approach.

(Slide.) This is the x-ray of such a patient, and the next slide shows the groove that may be produced by the subclavian artery. Since these were benign tumors I split the capsule and along a septum and removed the tumor in that manner from around the subclavian artery.

(Slide.) This is a case of sympathicoblastoma that presented the unusual initial finding of a massive hemothorax. This film was taken after the hemothorax had cleared and, at that time, the etiology of the hemothorax was unknown as the tumor was not visualized on the roentgenogram. Some months later (slide) one could see a small round mass

in the diaphragmatic region, and the lateral film shows the tumor to be somewhat further forward than most neurogenic tumors. As Dr. Pool mentioned, the sympathicoblastoma is a malignant tumor. This particular patient is well after two years.

DR. EARLE B. KAY, Cleveland.—I should like to discuss the technical aspects of operation of two types of neurofibroma; the cirsoid or plexiform neurofibroma and the dumbbell type of ganglionated neurofibroma that appears in children.

(Slide.) This demonstrates the extensive neurofibromatous infiltration in the apex of the left chest, with rib erosion, vertebral erosion, and the ribs separated. This was a 19-year-old boy. It was considered possibly malignant preoperatively, but it was known too that benign tumors could erode ribs as well.

(Slide.) The patient was explored through the fifth interspace. An extensive plexiform (cirsoid) neurofibroma was found infiltrating the brachial plexus, subclavian vessels, and the entire upper chest wall.

(Slide.) Some of these may be very extensive. It is a question how radical the operation should be. In this instance the entire upper five ribs were removed, leaving the subclavian vessels and brachial plexus along with the overlying muscles.

(Slide.) Next is the question of closure with such a large residual defect of the chest wall. The scapula bridged over the large defect. Only the muscles and soft tissues were closed. There has been no significant resulting defect.

(Slide.) This illustrates an example of the dumbbell neurofibromas seen in children. They are usually seen as a result of being asked by the orthopedist or neurosurgeon in consultation, because of spastic paraplegia. The question arises as to how they should be attacked. Should they have a laminectomy and the interdural aspect of the tumor removed first, or should they have a thoracotomy and the interpleural aspect removed?

(Slide.) In two cases laminectomy was performed first and the intradural aspect was removed; subsequently, after about two weeks, the intrapleural part was removed. At that time it is difficult to ascertain what is tumor and what is perhaps scar tissue. In the last two cases the thoracotomy has been the initial procedure, with removal of the intrapleural tumor. The spinal foramina can then be enlarged, or the intervening bone can be removed to allow removal of the intradural projection of the tumor. I think probably in most cases the tumor can be more effectively treated in this manner.

DR. DE WITT C. DAUGHTRY, Miami.—This is an interesting topic and I should like to show a couple of cases to illustrate that these lesions do not fall into a definite pattern, as you have already seen. Out of 13 cases, there were 2 with lesions extending through the foramina and compressing the spinal cord.

(Slide.) This roentgenogram shows multiple ganglioneuromas, two on the right and three on the left.

(Slide.) This is another roentgenogram of the same patient, showing three lesions on the left, two on the opposite side, and one below the diaphragm.

(Slide.) This is the roentgenogram of another patient, which shows in the apex of the right thorax a ganglioneuroma producing a considerable amount of pain for the size of the lesion. Upon exploration it proved to be a well-encapsulated, vascular lesion which seemed to pulsate when palpated. It was thought that this might be an aneurysm, and a needle was placed into it and 20 c.c. of bright red blood were aspirated. It was further freed and found to be not an aneurysm but a ganglioneuroma.

(Slide.) On cut section you can see the hemangiomatous type center, which gave the mistaken idea that it might possibly be an aneurysm.

In none of the 13 cases mentioned was malignancy found.

DR. POOL (closing).—I should like to thank the discussors, and to mention one point. In the group with ganglioneuromas, 8 patients were operated upon; 5 had partial excision. We felt that in these benign lesions partial excision was much safer where the lesion crossed the midline or was too extensive between the ribs. None of those tumors have shown further growth in the ensuing follow-up period—the longest period being four years. Whether partial excision is safe in a ganglioneuroma, only further time will tell.

TUMORS OF THE THYMUS

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O. THERON CLAGETT, M.D.,‡ AND (BY INVITATION) C. ALLEN GOOD, M.D.§
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A NUMBER of factors lend fascination and serve to heighten interest in the study of tumors of the thymus. Perhaps of first importance in this regard are the mysteries of origin of the thymic elements, the mysteries of the function of the gland, and the mysteries of the general alterations of the gland in disease. Likewise, interest comes from the obscure and tantalizing association of tumors of the thymus with myasthenia gravis, an association well known to us all, but understood by no one. It is hard to dismiss the occurrence of thymic tumors in 15 per cent of patients with myasthenia gravis[1] as simple coincidence. Nevertheless, little progress has been made in our understanding of this association during the past forty-nine years since Weigert[2] first reported a case of myasthenia gravis associated with a thymic tumor. Finally, of absorbing interest are unique clinical and pathologic features of the thymomas, a group of tumors about which much confusion and controversy center.

The controversies concerning classification and the variety of classifications that have been proposed for neoplasms of the thymus are the natural sequel of the incomplete knowledge of the origin of the cellular elements of the normal thymic parenchyma, and the common mistake of assigning a thymic origin to all tumors which are located in the anterior mediastinum. Since the small round cells of the thymus are morphologically identical with lymphocytes, the differentiation of tumors of the thymus arising from or composed almost completely of these cells, from tumors arising from lymph nodes in the anterior mediastinum may be difficult. The presence of these cells in large numbers in many thymomas has led to the erroneous idea that these tumors are lymphoblastomas. Conversely, because these small cells are known to exist in the normal thymus and in tumors of the thymus, lymphoblastomas arising in the anterior mediastinum have been erroneously classified as thymic tumors. Some of the confusion in classification has been the result of the unwillingness of some writers to classify the small thymocytes as lymphocytes because they believe that the thymocytes have an epithelial origin,[3] although most workers believe that they are lymphocytes[4] which have wandered into the gland.[5]

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PROBLEMS OF CLASSIFICATION

The problems involved in the classification of these tumors include not only those we have just mentioned, which have complicated the embryologic and histologic study of the gland, but those which arise from the fact that the anterior mediastinum, the seat of the thymus, contains lymph nodes which commonly are involved by primary or secondary neoplasms. Moreover, the anterior mediastinum is a common location for teratomas, perhaps the commonest tumors of this region. Added difficulties arise from the comparative rarity of thymic tumors and from the fact that other tumors of the anterior mediastinum, and for that matter, primary tumors in the lung,[6, 7] may secondarily involve the thymus and the thymic region by direct extension. The small-cell or oat-cell bronchiogenic carcinomas are particularly likely to involve the mediastinum and the small-cell pattern almost certainly has been mistaken for that of a malignant thymic tumor. Confusion may result from the fact that the thymus can participate in conditions in which the general lymphatic system is involved, such as leucemia[8]; however, most classifications exclude by definition such affections of the thymus. Finally, extensive degenerative changes within the tumor may make its identification on a histologic basis difficult or impossible.[9, 10]

These problems of classification apply to tumors which have their origin in the parenchyma of the thymus, and they are formidable. Nothing is to be gained by the attempt to recognize as thymic tumors those neoplasms which might arise in the stroma of the gland. Such tumors as fibrosarcoma, myxosarcoma, or lipoma which have been described as of thymic origin may occupy the region of the thymus without necessarily having arisen from the thymus.

THYMIC TUMORS IN MYASTHENIA GRAVIS

A benign character for tumors of the thymus associated with myasthenia gravis has been asserted by many writers,[8, 9, 11] and the case reported by Meggendorfer,[12] in 1908, in which a malignant thymic tumor was associated with myasthenia was cited as late as 1939[9] as being the only known exception. Recently, however, the reports of Murray and McDonald,[10] Blades,[13] Clagett and Eaton,[14] Keynes,[15] and Reid and Marcus[16] establish the invasive character of some of these tumors. Contrarily, it has been stated repeatedly that in the absence of myasthenia gravis thymic tumors are usually malignant and capable of widespread metastasis.[17-20] This is not in keeping with our concept which is based on the material that will be presented in a later section.

PERPLEXING ASPECTS OF THE ASSOCIATION OF MYASTHENIA GRAVIS WITH THYMIC TUMORS

Not only is it true in our experience that only 15 per cent of the patients with myasthenia gravis have thymic tumors, but in the group of patients who have had thymic tumors removed no uniform effect has been observed on the subsequent course of the myasthenia gravis.[1, 14, 15, 21] Furthermore, the time of appearance of the thymic tumor does not always coincide with the appearance of symptoms of myasthenia gravis.[1] Likewise, attempts to demonstrate a curare-

like substance in the extracts from several of the thymic tumors have failed.[1] These facts make a causal relationship between the thymic tumor and myasthenia gravis highly unlikely.

THYMIC TUMORS IN THE ABSENCE OF MYASTHENIA GRAVIS

Attention has been called to the opinions expressed by numerous workers[8-11, 22] that the thymic tumors occurring in patients with myasthenia gravis fall into a distinct group. We are in agreement with this point of view and, until rather recently, we held the corollary belief that this type of tumor occurred only in the patient with myasthenia. Now, however, we know that this latter view is incorrect[23, 24]; 11 of the 45 tumors in our series, to be reported on in a subsequent section, occurred in patients who had no evidence of having myasthenia gravis at the time of the operation and who subsequently have had no symptoms of this disease.

Most of the tumors that have been classified as thymic ones unassociated with myasthenia gravis have been highly malignant, and the sources of error attending the accurate classification of these lesions anatomically has been discussed at length. Perhaps some of the highly malignant tumors of the anterior mediastinum which have been classified as thymic tumors are actually thymic in origin, but in most of the reported cases the proof of their thymic origin is not convincing. Such are most of the cases reported by Lowenhaupt[20] who, in arriving at the group of tumors which she classified as thymic, included all anterior mediastinal tumors except those that were obviously metastatic to this region or that were a part of a generalized lymphoid disease. Since she apparently made no attempt to exclude malignant teratomas, acceptance of her interpretation and classification is dangerous. Not only could some of the tumors she included have been teratomas, but from the data she published, bronchiogenic carcinoma cannot be excluded definitely.

DEFINITION OF THYMOMA

For the only tumors which we believe can be designated as definitely thymic in origin, the term "thymoma" seems appropriate. A definition for the term "thymoma," which was first suggested by Grandhomme,[25] has rarely been attempted. As a generic term indicating the primary origin of the neoplasm from the thymic parenchyma and implying a lack of exact knowledge of the nature of the cellular elements from which it has arisen, it has much to recommend it.[17] Our definition is similar to that of Bell[8] and of Lièvre[22]: It is a slowly growing tumor of the thymus, which has arisen from both the epithelial (reticulum) and thymocytic elements of the thymic parenchyma. The relative proportion of these cells varies greatly from tumor to tumor and often from place to place in the same tumor. Typical Hassall's corpuscles are absent in most thymomas. Very common are such features as a dense fibrous capsule; distinct fibrous trabeculae; the palisading of epithelial cells about cystic spaces, about blood vessels, and about fibrous trabeculae; foci of necrosis, of cyst formation, and of calcification; but a number of tumors lack one or more of these features. All, however, contain the thymic lymphocyte or thymocyte and the thymic epithelial cell in varying proportions.

PLAN OF THE PRESENT STUDY

For this study, the records of those patients who registered at the Mayo Clinic during the period 1935 through 1949 and who had intrathoracic tumors of possible or probable thymic origin were reviewed. Only those cases in which tissue had been made available by surgical operation or by post-mortem examination were accepted for study; in this group, 45 patients had tumors which we classified as thymomas.

These cases of thymoma fell readily into two major groups clinically: those associated with myasthenia gravis and those unassociated with myasthenia gravis. Since, in our experience, 15 per cent of patients who have myasthenia gravis have a thymoma and 75.6 per cent of the patients with thymoma have myasthenia gravis, the division of our material on this basis for comparative analysis seemed sensible and challenging.

ANALYSIS OF CLINICAL DATA

In this small number of patients no essential difference in age could be made out in the two groups (Table I). By reason of the fact that approximately one-half of the patients with myasthenia gravis who come to the clinic are less than 40 years of age,[14] the occurrence of 22 (65 per cent) of the thymomas in association with myasthenia gravis in patients older than 40 years is probably significant.

TABLE I. CLINICAL FEATURES OF THYMOMA

CLINICAL DATA	PATIENTS WITH THYMOMA: MYASTHENIA GRAVIS	
	PRESENT (34)	ABSENT (11)
Age in years		
Extremes	26 to 68	16 to 65
Average	45.5	43.1
Sex		
Female	20	8
Male	14	3
Symptoms due to tumor		
Pain	4	4
Obstruction of superior vena cava	2	1*
Roentgenologic evidence of mediastinal tumor	33	11†

*This patient also experienced thoracic pain.
†Tumors of 7 of these patients were discovered by accident.

There was no significant difference in the sex incidence of thymomas in our series. In both groups of patients symptoms due to the presence of the tumor as such were exceptional. Since, with the exception of 1 tumor in this series, a thymoma is an anterior mediastinal lesion initially, it is not surprising that obstruction of the trachea or bronchi did not occur in any patient, or that signs of obstruction of the superior vena cava developed in three.

Of 34 patients with thymomas associated with myasthenia gravis, mediastinal tumors were demonstrated by roentgenologic means in thirty-three. In 1 patient the tumor was discovered only at the operation designed to remove the thymic gland in the hope that the course of the patient's myasthenia would be influenced favorably.

Pain in the chest was the result of involvement of the parietal layer of the pleura by the tumor. In view of the fact that complete surgical removal of the tumor was possible in only 3 of 8 patients who experienced pain in the thorax, the appearance of this symptom must be viewed with grave concern.

A possible relationship between hyperthyroidism and myasthenia gravis has been discussed by several writers.[26-28] With the exception of 1 patient with myasthenia who had a toxic adenomatous goiter, no endocrine disturbances were noted in any of our patients with thymomas.

ROENTGENOLOGIC DIAGNOSIS

There is nothing in the roentgenologic appearance of a thymoma to distinguish it from other tumors occurring in the anterior mediastinum. In all but 1 instance in our experience tumors of the thymus have been situated in the anterior portion of the mediastinum (Figs. 1 and 2). The 1 exception was a tumor found to lie posterior to the superior vena cava about midway between the sternum and the bodies of the thoracic vertebrae (Fig. 3). While the majority are situated anterior to the arch of the aorta and base of the heart this position is not constant; in our experience thymomas may be found at a level as low as the ninth or tenth thoracic vertebra, approaching the level of the diaphragm (Fig. 4).

The tumor is usually round or oval and sharply delineated from the surrounding tissue. In some instances, however, the mass may be somewhat flat and lie in close proximity to the anterior surface of the pericardium. In such cases the tumor appears as a bulge projecting forward from the cardiac shadow into the anterior mediastinum.

Sufficient calcium to be apparent roentgenologically was present in 13 of the 45 thymic tumors in our series (Fig. 4). It usually occupied a peripheral position, as though it were deposited in the wall of a cyst, but rarely it was distributed unevenly through the tumor, and in 1 case it seemed to be deposited in the central portion of the tumor. The presence of calcium in such quantities does not mean that the lesion is an innocent one, for in 1 case in which the tumor showed roentgenologic evidence of calcification, implants of tumor tissue were found on the pleural surface of the lung and diaphragm and on the posterior surface of the sternum. In another similar case the tumor had surrounded and obstructed the superior vena cava and had invaded the mediastinal pleura.

In 1947, one of us (Good[29]) reported on the roentgenologic recognition of thymic tumors in patients with myasthenia gravis. At that time it was noted that the ordinary posteroanterior roentgenogram of the thorax often failed to reveal a thymoma which subsequently could be found by roentgenoscopy or a roentgenogram made in the lateral projection (Fig. 1). Because of this fact all patients with myasthenia gravis seen at our institution are examined by means of roentgenoscopy and lateral roentgenograms, as well as the more routine posteroanterior stereoscopic pair. In a series of 249 consecutive patients with myasthenia gravis so examined, 37 tumors were found, an incidence of about 15 per cent.[30] Approximately 40 per cent of these tumors were not apparent

on the roentgenogram made in the frontal projection. It is our opinion that all patients known to have, or suspected of having, myasthenia gravis should receive the benefit of the more detailed roentgenologic examination outlined previously, because of the observed incidence of thymic tumors in this disease.

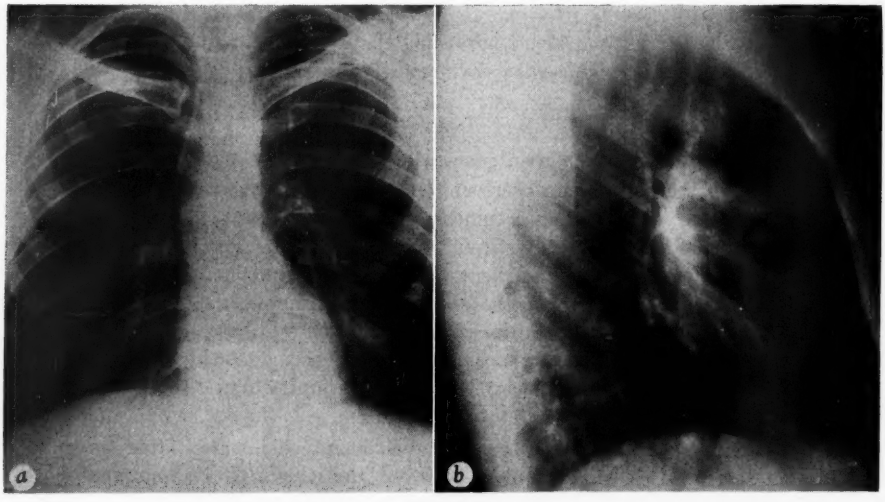

Fig. 1.—*a*, Posteroanterior view shows normal mediastinal shadow. *b*, Lateral view demonstrates a definite shadow in the anterior mediastinum at the level of the aortic arch.

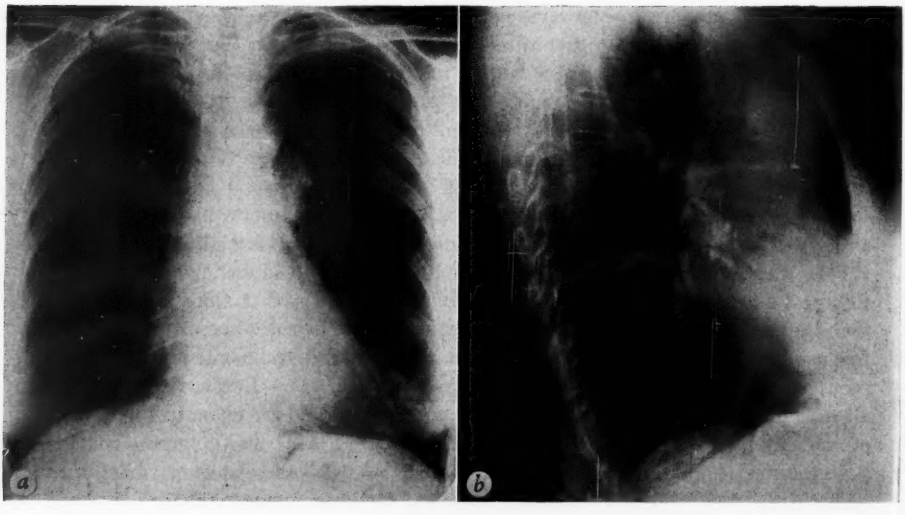

Fig. 2, *a* and *b*.—Anterior superior mediastinal tumor which proved to be a thymoma.

Although the roentgenologic appearance of a thymic tumor is not sufficiently characteristic to allow it to be distinguished from other tumors of the anterior mediastinum, the presence of a tumor in this location in a patient with myasthenia gravis makes a diagnosis of thymoma almost certain. We have never encountered a tumor in the anterior mediastinum of a patient with myasthenia gravis which proved to be anything other than a thymoma.

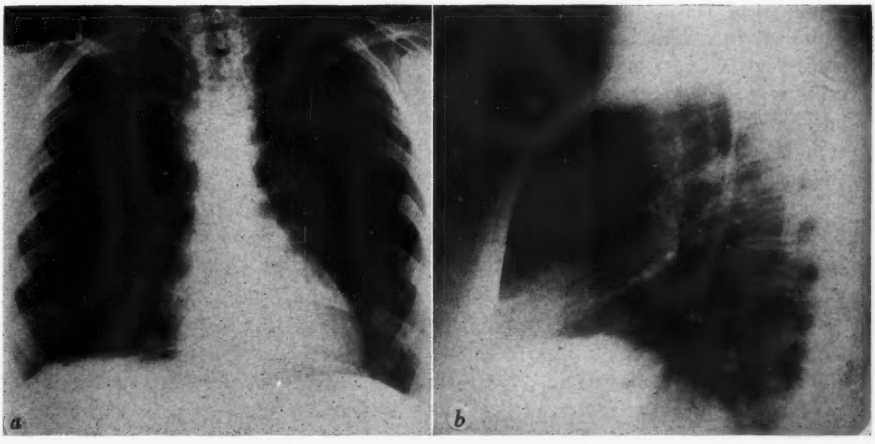

Fig. 3, *a* and *b*.—The shadow of a tumor which proved to be a thymoma can be seen in the middle and posterior portion of the superior mediastinum.

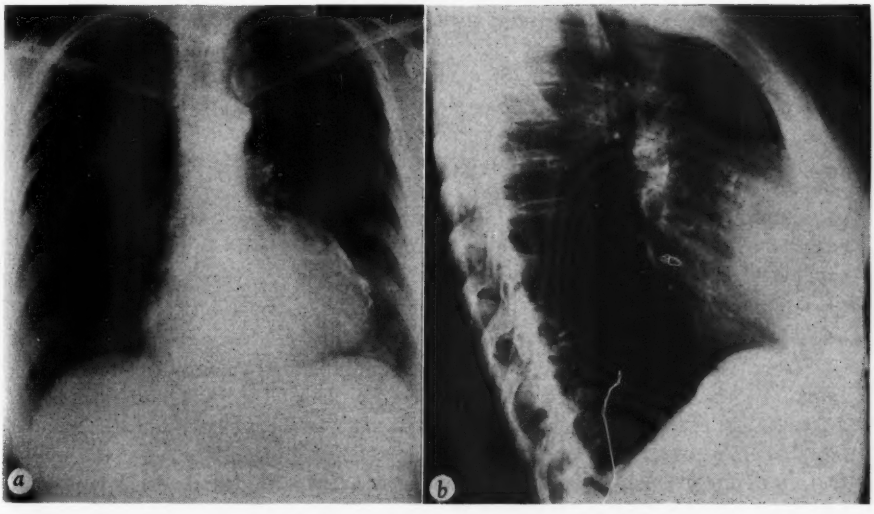

Fig. 4, *a* and *b*.—Anterior inferior mediastinal tumor which proved to be an encapsulated thymoma. Calcium is present.

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PATHOLOGIC CHARACTERISTICS

We believe that much of the past and present confusion in the classification of tumors of the thymus has been due to the inclusion, by a number of authors, of tumors that were not primary in the thymus but which arose primarily in the lung as bronchiogenic carcinoma, which were mediastinal manifestations of a generalized disease of the lymphatic system, or which were mediastinal teratomas. The perusal of case reports in the older literature leaves little doubt in the mind of the modern reader who is familiar with the pathology of bronchiogenic carcinoma that many of the latter of the small-cell or oat-cell type have been classified as malignant thymic tumors. Specifically we would mention the cases reported by Symmers[18] in 1932, most of which we would interpret differently today. Study of his case abstracts reveals that of the 8 patients whose tumors were called "peritheliomas," 6 were middle-aged men whose illness at the onset was characterized by respiratory symptoms: cough, "grippe," hemoptysis, and dyspnea. Extensive involvement of a lung and major or minor bronchi was described in 6 patients. It seems probable to us that these patients were victims of bronchiogenic carcinomas of the small-cell variety rather than of malignant tumors that arose in the thymus.

Our concept of the primary thymic tumors limits the group to those mediastinal tumors which are characterized histologically by the presence of both thymic epithelial (reticulum) cells and thymocytes in varying proportion. Such a concept not only fits our own material, but it provides a basis for understanding why some of the true thymomas have been described as lymphosarcomas and others as epitheliomas. The designation for the tumors in which the small round cell predominates almost to the exclusion of the epithelial cell has commonly been that of lymphosarcoma,[3] while for those tumors in which the reverse proportion exists, the diagnosis has usually been carcinoma or epithelioma.

On the basis of gross or microscopic examination the thymoma which has been removed from the patient with myasthenia gravis cannot be distinguished from the one which has been removed from the patient who is unaffected by this malady; but, while there is uniformity within wide but well-defined limits in the histologic pattern of all these thymomas, there is a great variation in the gross character of the tumors, some being solid, some being partially or almost completely cystic, the majority being encapsulated, but some being invasive and capable of surface implantation.

Deposits of calcium are fairly common in thymomas, both in the encapsulated and in the invasive and implanting varieties.

Gross Appearance.—(Fig. 5.) Grossly, a typical thymoma is a lobulated tumor with a thick, fibrous capsule which is complete (Fig. 5, *a*), but in almost a fourth of the tumors in our study the capsule was incomplete, and the tumor had invaded contiguous structures in the mediastinum. The most striking feature of the grayish-pink, cut surface of the average thymoma was lobulation produced by fibrous trabeculae (Fig. 5, *c*). Not all tumors, however, were lobulated (Fig. 5, *b*).

Cystic changes were present in most of the tumors (Fig. 5, *d*). The size and the number of cysts varied greatly, and in 3 instances no viable tumor cells could be identified in the walls of completely cystic thymic tumors removed from patients with myasthenia gravis. This complete absence of neoplastic tissue made absolute determination of the nature of the tumors impossible, but we feel that by inference they can be and should be grouped with thymomas. The preparation and study of many sections from 1 tumor that appeared to

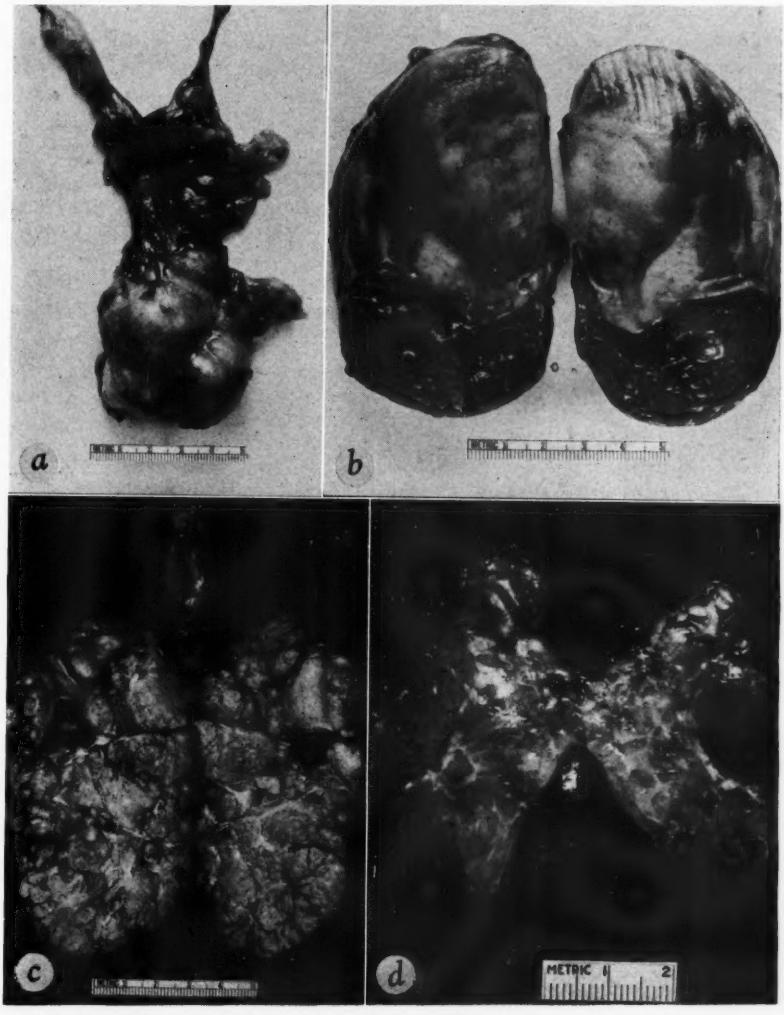

Fig. 5.—*a*, Thymus and thymoma from a patient with myasthenia gravis. *b*, Thymoma unassociated with myasthenia gravis. The capsule and a hemorrhagic cyst can be seen. *c*, Thymoma unassociated with myasthenia gravis. Lobulation and fibrous trabeculation are evident. *d*, Thymoma showing extensive cystic change. The patient had myasthenia gravis.

be completely cystic permitted a histologic diagnosis of thymoma. These cysts often contained a thick whitish material having the consistency of putty; less often they contained blood. Particularly in the tumors which were cystic, calcium deposits in the capsule and trabeculae were common.

Histopathologic Aspects.—The constant histologic feature of this group of 42 thymomas, excluding the 3 in which no viable neoplasm could be found, was the presence of the two cell types of the normal thymic parenchyma: the thymocyte and the epithelial cell (Fig. 6). The thymocyte is morphologically identical with the mature lymphocyte. The epithelial cell has often been called a reticulum cell in the thymus because of its similarity to reticulum cells in other organs. These epithelial cells usually have a distinct, pale nucleus with small nucleoli, varying amounts of cytoplasm, and indistinct cell membranes. In some of the tumors of our series epithelial cells had nuclei which were small and dark stained; in others these cells had foamy cytoplasm. Although the presence of both thymocytic and epithelial cells was invariable, the relative proportion of these cells varied widely from tumor to tumor and from place to place in the same tumor.

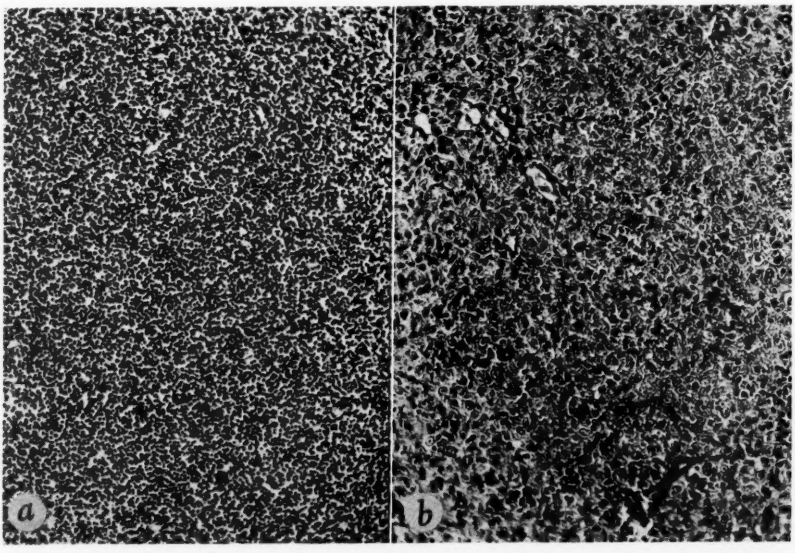

Fig. 6.—Thymoma (hematoxylin and eosin ×100). *a*, The thymocyte is the dominant cell in this tumor. *b*, The epithelial cell is the dominant cell.

Next in the order of importance as a distinctive histologic feature of thymomas was a palisaded arrangement of the epithelial cells about spaces, vessels, and fibrous trabeculae. This feature was present in 25 cases. A secretory function for the epithelial cells lining the cystic spaces was suggested to us by the presence of an acellular fluid in many of the spaces. This concept is consistent with the generally accepted idea that these cells are of an epithelial

nature. In parts of the tumor containing many of these adenomatoid structures, lumina of many sizes could be seen; some of these did not contain fluid (Fig. 7, *a* and *b*), while others did (Fig. 7, *c*). Hemorrhagic material in some cysts was undergoing organization (Fig. 7, *d*).

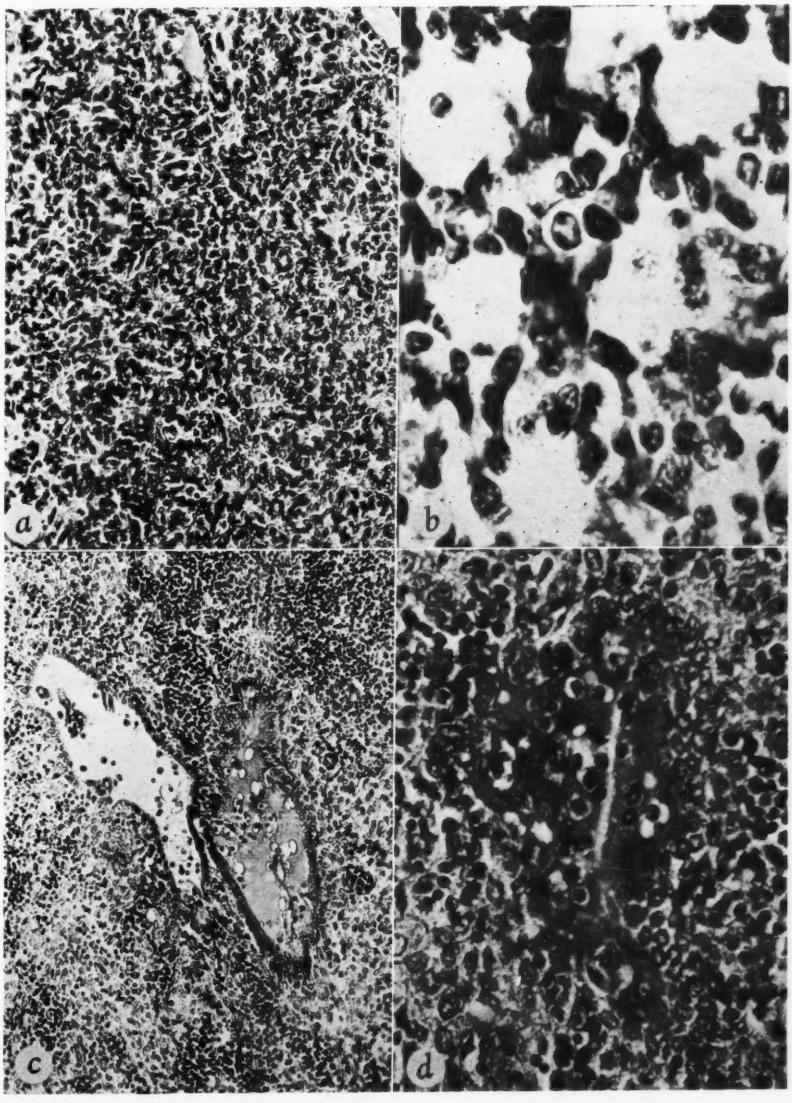

Fig. 7.—Thymoma (stained with hematoxylin and eosin). *a* and *b*, The adenomatoid arrangement of the epithelial cells is apparent. (*a*, ×165; *b*, ×880.) *c*, The epithelial cells have formed two "acini." The fluid in the one on the right is hemorrhagic (×100). *d*, The hemorrhagic fluid in the lumen of an "acinus" is organizing and in the left part of the acinus a small capillary is evident (×365).

The occurrence of these epithelial cells in palisade arrangement about spaces, about blood vessels imbedded in loose areolar tissue (Fig. 8, *a*), and about fibrous trabeculae (Fig. 8, *b*) suggested to us the possibility that these three features represent successive stages in a single process which begins with organization within the space. Perhaps hemorrhage which is rather frequent in these spaces provides the soil for the process of organization. Although palisading

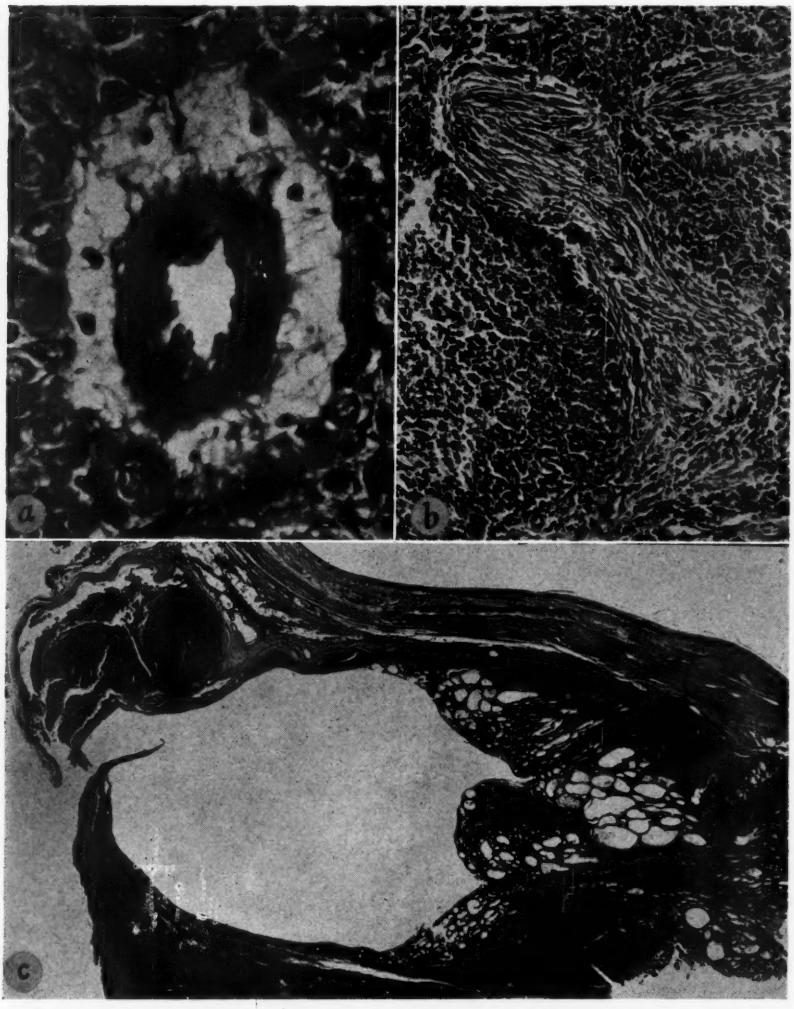

Fig. 8.—Thymoma. Stained with hematoxylin and eosin. *a*, Vessel surrounded by areolar tissue with palisaded epithelial cells (×500). *b*, Fibrous trabecula with the epithelial cells palisaded at right angles to the long axis of the trabecula (×165). *c*, Typical thick fibrous capsule and cystic spaces, some of them small, some quite large, lined by epithelial cells (×5).

was a frequent phenomenon about fibrous trabeculae it was not constant in our series.

Gross cystic changes (Fig. 8, *c*) in thymomas can be accounted in two ways: (1) on the basis of the progressive accumulation of fluid in these adenomatoid spaces either as a result of secretion or hemorrhage and (2) as a result of infarction with resulting liquefaction necrosis.

Hassall's corpuscles, the most characteristic feature of the adult human thymus, were present in only 11 of the thymomas in our series. A number of the structures which we called immature Hassall's corpuscles were whorls of epithelial cells (Fig. 9, *a*), but more often they were typical in appearance and a number were calcified (Fig. 9, *b*).

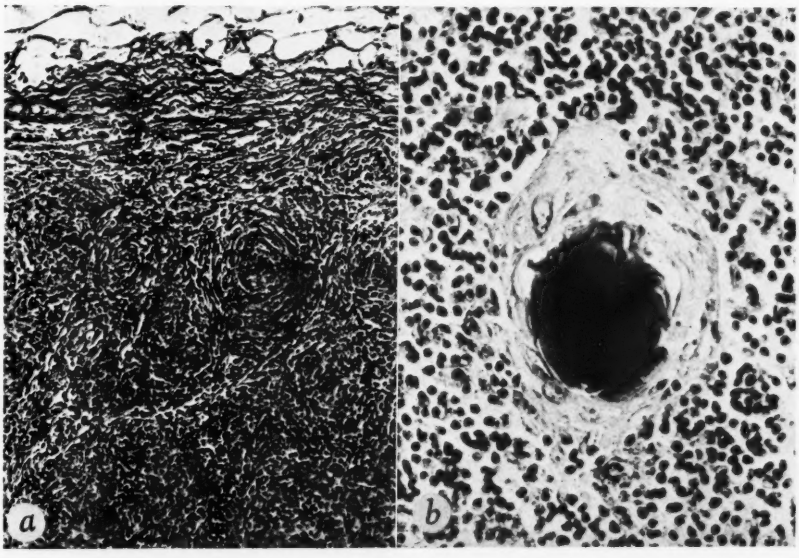

Fig. 9.—Thymoma. Stained with hematoxylin and eosin. *a*, Immature Hassall's corpuscles (×150). *b*, Mature Hassall's corpuscles with calcification (×365).

In 1 encapsulated thymoma the cell pattern was characteristic except for the presence of cells with multilobulated nuclei and pink-staining nucleoli that resembled Reed-Sternberg cells of Hodgkin's disease (Fig. 10, *a*). It seems likely that these were only variants of the epithelial cells.

The structure of the secondary deposits from the 2 tumors which spread by implantation to pleural surfaces was almost identical with that of the primary tumor; fibrous trabeculae were present and the same proportion of the two essential cells was preserved (Fig. 10, *b* and *c*).

Generally speaking, histologic features of malignancy were absent. In all tumors the epithelial cells had sharp perinuclear membranes; they were regular in size and showed little hyperchromasia. Rare mitotic figures were seen in

only a few tumors. Foam cells and cholesterol crystals were present, particularly in necrotic and degenerated sites. The thymocytic cells in all tumors resembled adult lymphocytes.

Tumor Behavior.—In several respects the behavior of these tumors is unusual. While most tumors which extend by invasion, also metastasize by the blood or lymph stream, thymomas seem to be an exception. Although in our series 10 of 45 tumors invaded contiguous structures and 2 spread to pleural

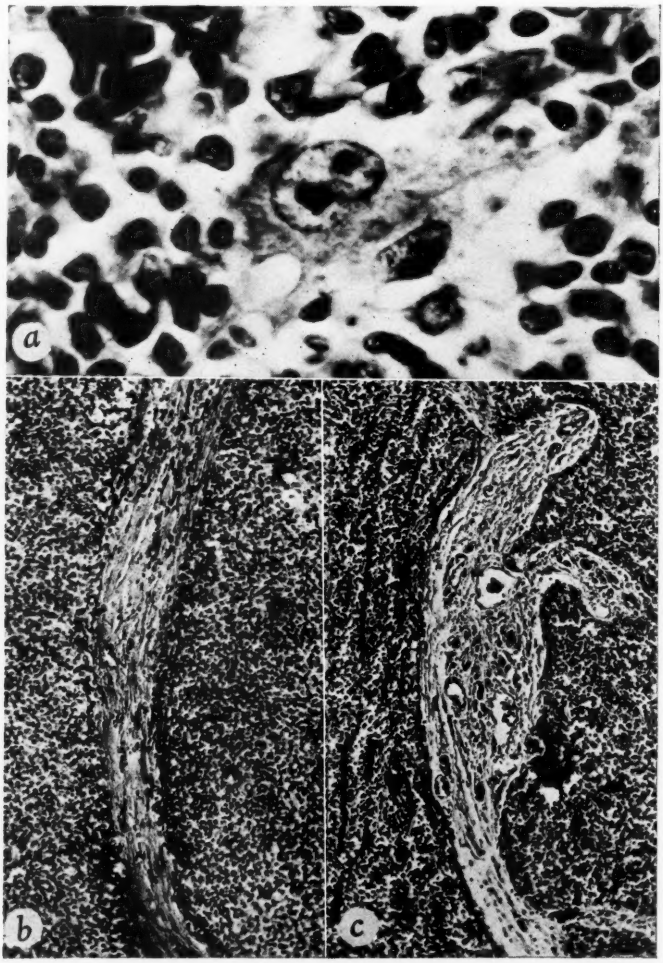

Fig. 10.—Thymoma. Stained with hematoxylin and eosin. *a*, Cell with a multilobulated nucleus that resembles a Reed-Sternberg cell (×1,040). Such cells were present in only one tumor. *b*, Primary tumor demonstrating fibrous trabeculae and the mixture of thymocytic and epithelial cells (×100). *c*, Pleural implant showing a histologic pattern similar to that of the primary tumor (×100).

TABLE II. MODE OF EXTENSION OF THYMOMA IN 11 CASES

	CASES
Invasion of	
Pleura	6
Pericardium	5
Superior vena cava and innominate veins	5
Lung	3*
Implantation to parietal and visceral pleura	2

*Only the superficial aspects of the lobe adjacent to the mediastinal tumor were involved.

surfaces by implantation, none produced embolic metastasis (Table II). The histologic appearance of these tumors which had extended to other structures differed in no particular from that of the tumors which were completely encapsulated. Furthermore, the tumors of the patients who had myasthenia gravis seemed to behave exactly like those which were unassociated with myasthenia.

The tendency of these tumors to extend by invasion and by implantation has been described by previous authors.[10, 12-16] Obstruction of the superior vena cava and the innominate veins is the result of compression by the tumor in some patients, but actual invasion of the venous wall does occur. We have been unable, however, to find evidence of extension of the tumor into the lumen of the vein. Since most of these tumors lie in contact with the pericardium, it is not surprising to find that it is involved frequently by the invasive tumors. One tumor had extended through the fibrous pericardium to form a mass within the pericardial sac.

Complete necropsies were done in 11 cases. In none was there evidence of lymphogenous or hematogenous metastasis. It is worth mentioning that lymphorrhages which are common in the skeletal muscle of patients with myasthenia gravis may be mistaken for metastatic lesions. Such a mistake accounts for some of the reports of embolic metastasis that are to be found in the literature.

Rate of Growth—There is much evidence that thymomas are slow-growing tumors, both in the patients who have myasthenia gravis and in those who do not. In our series, of the patients in the former group 1 was known to have a mediastinal tumor ten years, and another five and one-half years prior to

TABLE III. FATE OF PATIENTS WITH INERADICABLE THYMOMA

	PATIENTS WITH THYMOMA: MYASTHENIA GRAVIS	
PRESENT STATUS	PRESENT	ABSENT
Dead	5	0
Died while in hospital	2	0
Died since dismissal	3	0
Alive	1	3
Condition satisfactory	1*	1†
Condition worse	0	2‡
Lost to follow-up	0	1§
Total	6	4

*Fifty months after operation.
†Six months after operation.
‡Each patient survived nineteen months after operation.
§This patient had an ineradicable lesion in 1946 and, letters of inquiry remaining unanswered, is presumably dead.

TABLE IV. SURGICAL TREATMENT OF THYMOMA: FORTY-ONE PATIENTS*

	PATIENTS WITH THYMOMA: MYASTHENIA GRAVIS	
	PRESENT	ABSENT
Tumors removed surgically	24	7
Died while in the hospital	2	1
Cause of death:		
Myasthenia gravis	2	
Pulmonary complications		1
Ineradicable tumors explored surgically: biopsy	6	4
Died while in the hospital	2	0
Cause of death:		
Myasthenia gravis	1	
Rupture of the ascending aorta	1	

*In 4 additional cases of thymoma associated with myasthenia gravis, the tumor was not explored surgically but was removed at necropsy.

operation. In the latter group, 2 were known to have had their tumors four and six years, respectively, before operation. Additional evidence of slow progression of thymomas is presented in Table III where it can be seen that of the 4 patients with ineradicable thymomas who are known to be alive, 1 has survived six months, 2 nineteen months, and 1 fifty months since operation. The duration of survival after operation for the 3 patients who had ineradicable lesions and who died after dismissal from the hospital was seventeen, forty-one and fifty-one months, respectively.

SURGICAL TREATMENT

The selection by the surgeon of the proper operative approach to a thymic tumor should be based upon a consideration of the size and location of the tumor.[15, 31, 32] For a large tumor that presents chiefly in one side of the thoracic cavity, a posterolateral, transpleural approach allows the surgeon ready access. This approach will permit the removal of large tumors that could not be easily removed otherwise. For small tumors that lie essentially in the midline, we prefer a sternum-splitting incision which provides excellent exposure of all the structures in the anterior portion of the mediastinum, and it does not necessitate entry of the pleural space. The latter advantage is shared by the transcostal or parasternal approach, but this advantage is more than offset by the inadequacy of the surgical exposure which the parasternal approach provides.

The surgical procedures carried out on the 41 patients included in our study were of three classes: removal of the tumor, removal of the tumor and the thymus, and biopsy or only partial removal of the tumor. In our experience

TABLE V. RESULTS OF THE SURGICAL REMOVAL OF THYMOMA

PRESENT STATUS	PATIENTS WITH THYMOMA: MYASTHENIA GRAVIS	
	PRESENT	ABSENT
Dead	7	1
Died in the hospital after operation	2	1
Died since dismissal from the hospital	5	0
Alive: no evidence of recurrence or metastasis	16	6
Lost to follow-up	1	0
Total	24	7

TABLE VI. PRESENT STATUS OF PATIENTS WITH THYMOMA WHO WERE OPERATED ON FIVE OR MORE YEARS AGO

	PATIENTS
Alive without evidence of recurrence	7
Alive but with evidence of progression 51 months after operation, December, 1948	1
Dead	8
Lost to follow-up	1

removal of the entire thymus together with the tumor seems to offer no advantage over the removal of the tumor alone. In the group of patients whose thymoma could be removed completely, none have died because of local recurrence of, or distant metastasis from, the tumor after being dismissed from the hospital. The results of surgical treatment of these patients are summarized in Tables III through VII.

Of the 17 patients with thymomas who were operated upon five or more years ago (Table VI), 4 had ineradicable lesions; 3 of the 4 are dead, presumably as a direct result of the tumor. The fourth patient was alive when he was last heard from in December, 1948, fifty-one months after exploration, but his condition was poor because of the existence of obstruction of the superior vena cava.

TABLE VII. ANALYSIS OF THE CAUSES OF DEATH

TIME AND CAUSE OF DEATH	PATIENTS WITH THYMOMA: MYASTHENIA GRAVIS	
	PRESENT	ABSENT
Died in the hospital after operation	8	0
Cause: Myasthenia gravis	7	0
Rupture of the aorta	1	0
Pulmonary complications	0	1
Died after dismissal from the hospital	8	0
Cause: Myasthenia gravis	4	0
Brain tumor (astrocytoma)	1	0
Unknown	3*	0

*Since these 3 patients had ineradicable tumors, death most probably was caused by the tumor as such.

Removal of a thymoma from a patient with myasthenia gravis has no evident influence on the course of the disease.[1]

RESPONSE OF THYMOMAS TO ROENTGEN THERAPY

Information bearing on the response of thymomas to roentgen therapy was available in two forms: in the size of tumors after roentgen therapy as compared to their size before such treatment, and in the histologic appearance of those tumors which had been irradiated as compared to the appearance of those which had not. Data which would permit an estimate of change in size of a tumor after roentgen therapy were available on 12 patients who had myasthenia gravis; in 10 no change was apparent; in 1 who had an ineradicable thymoma the tumor became smaller and ultimately could not be demonstrated roentgenologically, but the patient died fifty-one months after operation, presumably as a result of the tumor; and in 1 the tumor, which was observed to become

smaller, was found at operation to be a fibrous-walled cyst. This latter change, however, could not with certainty be ascribed to roentgen therapy because another of our patients who had no roentgen therapy had a completely cystic lesion. Evidence of a similar nature from the patients who had thymoma unassociated with myasthenia gravis also failed to show any definite benefit to the patient from roentgen therapy.

Roentgen therapy in the doses employed for these patients had no appreciable effect upon the microscopic picture of the thymoma.

The report of Reid and Marcus[16] afforded evidence that some of these tumors are radiosensitive, that every patient with an ineradicable thymoma should be given intensive treatment.

SUMMARY AND CONCLUSIONS

Forty-five cases of thymoma in which tissue had been made available by surgical operation or by post-mortem examination form the basis of this report. We have defined a thymoma as a specific tumor of the thymus that is characterized by the presence of both thymic epithelial and thymocytic cells in varying proportions. Though this tumor is commonly associated with myasthenia gravis, only 15 per cent of the patients who have myasthenia have a thymoma, and 25 per cent of the tumors in our experience occurred in patients who had no evidence of myasthenia. For the purpose of comparative analysis the patients with thymomas were divided into two groups on the basis of the presence or absence of myasthenia. In both groups thymomas occurred almost exclusively in adults of both sexes, and symptoms due to the presence of the tumor as such were uncommon.

To attain the highest possible degree of accuracy in the roentgenologic diagnosis of thymomas posteroanterior and lateral roentgenograms and roentgenoscopy of the thorax should be employed in every patient with myasthenia gravis. Characteristically, these tumors occur in the anterior mediastinum in a position anterior to the aortic arch and the base of the heart. Some of them contain calcium in quantities sufficient to be apparent roentgenologically. Peculiar features which would permit a pathologic diagnosis on the basis of the roentgenologic findings were lacking.

Three-fourths of the thymomas in this series had a distinct fibrous capsule, but the others had broken through their capsule to invade adjacent structures: the pericardium, the pleura, the superior vena cava and the left innominate vein; and 2 spread to parietal and visceral pleural surfaces as nodular implants of tumor. No evidence was found to suggest that embolic lymphogenous or hematogenous metastasis occurred. We failed in our attempts to separate, on histologic grounds, those tumors which were invasive and those which spread by implantation, from those which were completely encapsulated.

The selection by the surgeon of the proper surgical approach should be based upon a consideration of the size and the location of the tumor.

Because of the poor general condition of 4 patients with myasthenia gravis, no surgical treatment was attempted. Of the other 41 patients, 31 underwent

complete removal of the tumor and 10 underwent incomplete removal or biopsy. The cause of death of 3 of the 5 patients who died in the hospital after operation was myasthenia gravis.

In no patient whose tumor was removed completely has there been evidence of recurrence. Most of the deaths have been due to myasthenia gravis and not to the tumor itself, although in the group with ineradicable lesions, the tumor itself was usually fatal.

Thymomas cannot be regarded as innocent. Because there is no basis clinically or roentgenologically for the accurate pathologic diagnosis of mediastinal tumors, exploratory thoracotomy is indicated for all these patients whose general condition will permit.

Intensive roentgen therapy for ineradicable tumors seemed to benefit some patients.

REFERENCES

1. Eaton, L. M., Clagett, O. T., Good, C. A., and McDonald, J. R.: Thymectomy in Treatment of Myasthenia Gravis; Report Based on Thirty-two Cases, Arch. Neurol. & Psychiat. **61**: 467-498, 1949.
2. Weigert, Carl: Pathologisch-anatomischer Beitrag zur Erb'schen Krankheit (Myasthenia gravis), Neurol. Centralbl. **20**: 597-601, 1901.
3. Ewing, James: Neoplastic Diseases; a Treatise on Tumors, ed. 4, Philadelphia, 1940, W. B. Saunders Company, 1160 pp.
4. Maximow, A. A., and Bloom, William: A Textbook of Histology, ed. 4, Philadelphia, 1942, W. B. Saunders Company, pp. 326-327.
5. Norris, E. H.: The Morphogenesis and Histogenesis of the Thymus Gland in Man: in Which the Origin of the Hassall's Corpuscles of the Human Thymus Is Discovered, Carnegie Institute, Contrib. Embryol. **27**: 191-207, 1938.
6. Willis, R. A.: Pathology of Tumours, St. Louis, 1948, The C. V. Mosby Company, 992 pp.
7. Blades, Brian: Relative Frequency and Site of Predilection of Intrathoracic Tumors, Am. J. Surg. **54**: 139-148, 1941.
8. Bell, E. T.: Tumors of the Thymus in Myasthenia Gravis, J. Nerv. & Ment. Dis. **45**: 130-143, 1917.
9. Blalock, Alfred, Mason, M. F., Morgan, H. J., and Riven, S. S.: Myasthenia Gravis and Tumors of the Thymic Region; Report of a Case in Which the Tumor Was Removed, Ann. Surg. **110**: 544-561, 1939.
10. Murray, N. A., and McDonald, J. R.: Tumors of the Thymus in Myasthenia Gravis, Am. J. Clin. Path. **15**: 87-94, 1945.
11. Norris, E. H.: The Thymoma and Thymic Hyperplasia in Myasthenia Gravis With Observations on the General Pathology, Am. J. Cancer **27**: 421-433, 1936.
12. Meggendorfer, F.: Quoted by Bell.[8]
13. Blades, Brian: Mediastinal Tumors; Report of Cases Treated at Army Thoracic Centers in the United States, Ann. Surg. **123**: 749-765, 1946.
14. Clagett, O. T., and Eaton, L. M.: Thymic Tumors in the Myasthenia Gravis, S. Clin. North America **23**: 1076-1082, 1943.
15. Keynes, Geoffrey: The Surgery of the Thymus Gland, Brit. J. Surg. **33**: 201-214, 1946.
16. Reid, Hugh, and Marcus, R.: Thymoma With a Report of 5 Cases, Brit. J. Surg. **36**: 271-279, 1949.
17. Margolis, H. M.: Tumors of the Thymus: Pathology, Classification and Report of Cases, Am. J. Cancer **15**: 2106-2142, 1931.
18. Symmers, Douglas: Malignant Tumors and Tumor-like Growths of the Thymic Region, Ann. Surg. **95**: 544-572, 1932.
19. Crosby, E. H.: Malignant Tumors of the Thymus Gland, Am. J. Cancer **16**: 461-486, 1932.
20. Lowenhaupt, Elizabeth: Tumors of the Thymus in Relation to the Thymic Epithelial Anlage, Cancer **1**: 547-563, 1948.
21. Blalock, Alfred: Thymectomy in the Treatment of Myasthenia Gravis; Report of Twenty Cases, J. THORACIC SURG. **13**: 316-339, 1944.
22. Lièvre, J. A.: Peut-on tenter un traitement chirurgical de la myasthénie? Presse méd. **1**: 991-992, 1936.
23. Andrus, W. D., and Foot, N. C.: Report of a Large Thymic Tumor Successfully Removed by Operation, J. THORACIC SURG. **6**: 648-659, 1937.

24. Heuer, G. J., and Andrus, W. D.: The Surgery of Mediastinal Tumors, Am. J. Surg. **50**: 146-224, 1940.
25. Grandhomme, F.: Quoted by Reid and Marcus.[16]
26. Cohen, S. J., and King, F. H.: Relation Between Myasthenia Gravis and Exophthalmic Goiter, Arch. Neurol. & Psychiat. **28**: 1338-1345, 1932.
27. Eaton, L. M.: Myasthenia Gravis: Its Treatment and Relation to the Thymus, Proc. Staff Meet., Mayo Clin. **17**: 81-87, 1942.
28. McEachern, Donald: The Thymus in Relation to Myasthenia Gravis, Medicine **22**: 1-25, 1943.
29. Good, C. A.: Roentgenologic Findings in Myasthenia Gravis Associated With Thymic Tumor, Am. J. Roentgenol. **57**: 305-312, 1947.
30. Good, C. A.: Unpublished data.
31. Clagett, O. T., and Root, G. T.: Surgical Approach for Tumors of Thymus, Surg., Gynec. & Obst. **78**: 397-401, 1944.
32. Clagett, O. T., Eaton, L. M., and Glover, R. P.: Thymectomy for Myasthenia Gravis; Surgical Technique, Surgery **26**: 852-860, 1949.

DISCUSSION

DR. MARK H. WILLIAMS, Binghamton, N. Y.—A thymoma may be an unexpected finding at operation. Because of the unusual x-ray findings and its deceptive gross appearance, it may be of interest to show an example of a benign asymptomatic tumor in this category, which we recently encountered in a 9-year-old boy. This tumor was discovered when an x-ray examination was made following a chest injury.

(Slide.) Among the numerous x-ray studies which were made, only these two films demonstrate a double shadow. This is seen along the right cardiac border in the postero-anterior view; it is superimposed on the heart shadow and is demonstrated to better advantage in the lateral view. Fluoroscopic examination was helpful in establishing a diagnosis. At the screen, pulsations were absent along the right border of the heart. Our preoperative diagnosis was mediastinal cyst. The tumor was approached by an incision through the right fifth interspace. At operation it appeared unquestionably to be an encapsulated lipoma; it extended over the heart anteriorly and upward over the great vessels to the thoracic inlet. After extending the incision upward and dividing the fifth and fourth costal cartilages, sufficient exposure was obtained to remove the tumor without difficulty.

(Slide.) The gross appearance of the tumor is typical of lipoma. It was much bulkier than one would expect from the x-ray findings; it weighed 255 grams.

(Slide.) The microscopic findings were surprising. Throughout this huge fatty tumor small islands of thymic tissue were found. When we went back to the gross specimen, these were readily seen as 4 or 5 mm. yellowish areas.

(Slide.) This is a recent follow-up on this boy, who was operated upon about nine months ago. He made a good recovery from the operation and has remained well.

DR. JOHN M. DORSEY, Chicago.—I should like to show slides of a patient upon whom we operated, who, I thought, had an anterior mediastinal tumor, perhaps a dermoid.

(Slide.) This shows the microscopic picture, as does the next. This tumor in the final analysis proved to be a thymoma, but I think it is well worth while to record our experience. In preoperative study, investigating diplopia as a possible symptom of myasthenia gravis with a therapeutic dose of neostigmine and finding no therapeutic response, we thought we had ruled out thymoma. The patient was given, unwittingly, curare along with pentothol during induction of anesthesia and developed respiratory arrest, necessitating postponement of surgery for a week. We should have come to the opinion immediately that this was a thymoma with latent myasthenia gravis. On removing the tumor the following week we did have a frozen section which revealed it to be a thymoma. The patient had a rather stormy postoperative course and died on the third postoperative day.

In summary, I feel that curare should be avoided in anesthesia for anterior mediastinal tumors. Immediate frozen section should be made. If they resemble tumors of thymic origin in any way, prostigmine and general supportive measures such as given myasthenics should be instituted.

DR. EDGAR W. DAVIS, Washington, D. C.—I was rather surprised to hear Dr. Seybold say, if I understood him correctly, that he could not find any definite relationship between the thymus and myasthenia gravis and that, in his opinion, the subsequent course of myasthenia gravis was not influenced by removal of the thymus gland. I should like to ask Dr. Seybold if he would care to comment on the diametrically opposite conclusions from the series of cases presented by him, which obviously have been beautifully studied, as compared with the 120 cases presented by Keynes of England. Out of Keynes' 120 cases, he reports 39 patients who are well, free from symptoms, and who do not need prostigmine, and 40 patients who are virtually well, with minimal symptoms. As I recall, Keynes' patients have been observed over a long period dating back to 1942.

DR. SEYBOLD (closing).—I should like to thank Dr. Williams, Dr. Dorsey, and Dr. Davis for their discussion. First, as to Dr. Dorsey's comments about keeping myasthenia gravis in mind in dealing with a patient who has a tumor which may be a thymoma. We have used provocative tests for myasthenia in a number of these patients. Sometimes the weakness complained of by some patients with tumor is difficult to differentiate from the specific weakness of myasthenia gravis, and it is indeed dangerous to operate on a patient with myasthenia gravis whose disease is unrecognized. It is dangerous unless it is previously recognized and the patient is adequately prepared and treated with prostigmine and other drugs that are effective.

The relation of thymic tumor to myasthenia gravis has received a lot of attention and, as we all know, within the last few years—about 1939, I believe—Dr. Blalock reported a patient on whom he had removed a thymic tumor three years before and in whom, at the time of his report, remission of the disease had followed. Since that time considerable experience has accumulated. We all know that myasthenia gravis is a remitting disease, and the efficacy of any form of treatment is difficult to assess, particularly on a short time basis. Quite recently, Dr. Eaton of our Neurological Section, who has been most interested in this problem, has published a follow-up on the patients he has been studying for eight years, and you can see from the figures I gave you in the charts that of the patients who had thymic tumors removed, approximately one-half of them are dead, and the leading cause of death was myasthenia gravis. I do not know how to account for the discrepancy between our experience which, I might add parenthetically, is in keeping with the experience of others throughout the country—the discrepancy between our experience and that of Dr. Keynes, except to say that some of these patients do seem to be benefitted for a time. But we know that myasthenia gravis, without treatment, will go into remission, and some of those remissions are essentially permanent in that they have lasted 15 or 20 years.

SYPHILIS OF THE ESOPHAGUS

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CHICAGO, ILL.

TERTIARY syphilis is a very occasional cause of obstruction of the esophagus. Being rare, it is important that every proved instance be recorded in the literature so that knowledge of the condition be increased and the profession be frequently reminded of its existence. The purpose of this paper is to report such a case and review the accumulated literature.

CASE REPORT

A 58-year-old white man was hospitalized because of difficult swallowing. Within a few days after admission he was regurgitating all food and liquids. He stated that for several years he had experienced periodic difficulties in swallowing food and liquids.

He complained of a choking sensation, tightness in the throat, and slight pain when swallowing. The laboratory examinations were not remarkable, save for highly positive Kahn and Wassermann reactions. X-ray study of the esophagus revealed a spherical tumor (Fig. 1) almost completely occluding the lumen at the cardic end. At esophagoscopy the lower end was found to be completely filled by a flesh-colored, finely irregular, nonulcerative mass. Little bleeding resulted from biopsy. Microscopic study of the specimen secured revealed only stratified squamous epithelium.

Because of the long history of dysphagia and the positive serology, it was felt worth while to give antisyphilitic therapy. The result was astounding. Relief from all substernal discomfort and from difficult swallowing was virtually complete in only a few days after the first injection of an arsenical. Continuation of the antisyphilitic treatment soon resulted in total relief of all symptoms.

Subsequent x-ray study of the esophagus (Fig. 2) showed disappearance of the tumor previously seen. Esophagoscopy was repeated sixty days after the first examination. No tumor and no abnormality were found. The mucosa at the cardiac end was normal in appearance.

The patient was discharged with instructions to continue the antisyphilitic treatment. Follow-up one year after discharge revealed that he had not received any treatment since discharge and within two months after leaving the hospital the symptoms had recurred. He did not return to the hospital.

REVIEW OF LITERATURE

Contributions to medical literature dealing with syphilis of the esophagus are rare. Guyot[1] in reviewing the literature dating from 1717 to 1930, from America, England, France, Germany, and Austro-Hungary [*sic*] found but fifty-five recorded instances. Moore,[2] in admitting that syphilis of the esophagus has a place in the literature, stated he had never personally observed a case, and that most of those patients thought to have syphilis of the esophagus were eventually found to have cancer. Guisez,[3] in over 3,000 esophagoscopies, saw only one instance of tertiary syphilis of the esophagus.

To the fifty-five cases found in the literature to 1930 by Guyot[1] he himself added two personal cases. Vezina,[4] Lukens and Ono,[5] Shimokawa,[6] Hanif,[8] Wilcox,[9] Fontana,[7] and Ponzoni[13] have each added reports of one case since 1930.

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Glass and Freeman[10] and Avery[11] have added two cases each. Watson-Williams[14] reported four cases. Sabalette and Martinez[15] and Postigo[16] have reported one case each.

With the case which forms the basis of this paper, a total of seventy-five cases of tertiary syphilis of the esophagus have been found in the literature, twenty having been recorded since 1930.

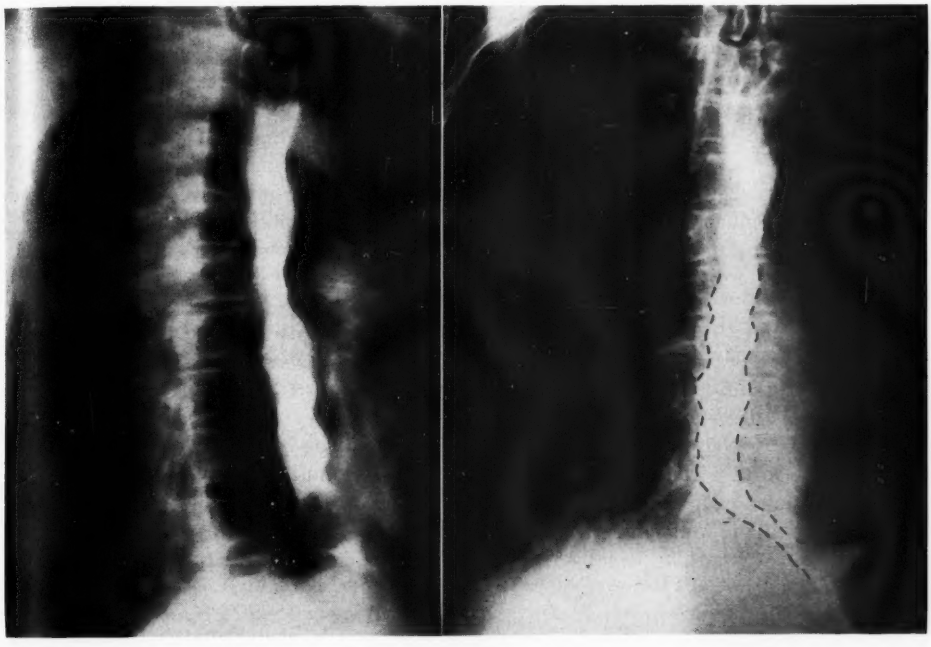

Fig. 1. Fig. 2.

Fig. 1.—Rounded tumor mass seen obstructing flow of barium in lower esophagus.

Fig. 2.—Esophagram taken two weeks after institution of antisyphilitic treatment. Tumor no longer present and barium flows freely into the stomach.

PATHOLOGY

The gross structure of the lesion of tertiary syphilis of the esophagus may assume one of two general forms, a submucous gumma or a diffuse inflammatory reaction with destruction of tissue and resultant scarring and stenosis. Either form may display erosions and ulcerations. The gummas apparently are prone to ulceration and loss of tissue with subsequent scarring. The microscopic findings have been variously described by the few having material at their disposal. In general, the picture is that of periarteritis leading to scar formation.

SYMPTOMS

In general, the symptoms are those of obstruction of the esophagus. In addition, there may be cough, expectoration, and hemoptysis. These are symptoms of tracheal perforation.

Guyot[1] mentioned the absence of pain in tertiary syphilis as a possible differential point between this disease and carcinoma of the esophagus. Four of the patients whose cases are reviewed complained of some sort of substernal distress. All the other reports do not mention the presence of pain or specifically say no pain was present. Wile[17] stated, "Pain is not a marked symptom except during the act of swallowing. When there is ulceration, pain may be present during repose as well as deglutition. In old standing cases where scarring and consequent narrowing of the tube occur, the pain during attempted swallowing may be very severe."

The length of time of the dysphagia appears to be a fairly reliable differential symptom. In those histories in which the duration was recorded the figures were—three years, eight years, seven years, several years, ten days, six weeks, three months, two months, four months, several weeks, and eight weeks. The longer periods will, of course, eliminate esophageal cancer in any questioned case. The apparent fact that the clinical course of tertiary syphilis of the esophagus is more prolonged than that of cancer was previously noted by Myerson.[12]

X-RAY EXAMINATION

Roentgen examination of the barium-filled esophagus is of great importance in the diagnosis of any obstructive lesion. Wilcox[9] has said that the roentgenoscopic appearance of tertiary syphilis of the esophagus is more or less characteristic. He described the walls of the tube as being very rigid with little change in contour or size of the barium column as the opaque bolus passes through the stenotic area. The barium has a finely irregular outline. This is in contradistinction to carcinoma of the esophagus which, if extensive enough to produce rigidity of the wall for a considerable distance, will produce coarse irregularities with more irregular canalization. In spasm of the esophagus the outline is smooth.

ESOPHAGOSCOPY

Avery[11] stated that very few descriptions of the endoscopic findings of tertiary syphilis of the esophagus are to be found in the literature. This phase of the examination is of the greatest importance and for this reason summaries of the endoscopic descriptions of cases as they appear in the literature are presented here.

Hanif's[8] examination recorded severe injection of the mucous membrane and multiple ulceration. Guyot[1] found the esophageal wall stiff and rigid with the mucous membrane stretched and adherent. Ulcerations with sharply demarcated edges and dirty sloughs at the bases were seen. Vezina[4] reported the esophagus of his patient to show that the mucous membrane covered the granulations which bled easily. A stenosis at the cardiac end could not be passed. Fontana[7] noted ulcerations with plaques of leucoplakia nearby. The mucous membrane appeared to be involved in a "nonspecific" esophagitis with hypertrophy of the wall and dilatation of the tube above the lesion. Myerson[12] described an umbrella-like convexity of the esophageal wall which appeared intensely red and slightly swollen. The lesion bled easily.

Watson-Williams[14] reported the endoscopic findings in four patients. In the first, there was a patch of pale leucoplakia seen 3 cm. inside the esophagus. The middle one-third showed a well-marked esophagitis with patches of pale, sodden epithelium, arranged in longitudinal streaks. His second patient showed dilatation of the upper part of the esophagus. Below 27 cm. the mucosa was thickened and macerated; at 30 cm. there was a longitudinal dull streaking of the walls, apparently from denudation; at 33 cm. bright scarlet patches began to appear devoid of epithelium and bleeding on contact. At 37 cm. the whole circumference was superficially ulcerated and so contracted that only a small lumen remained. His third patient had received antisyphilitic treatment before esophagoscopy. In this case a diffuse esophagitis without special features was seen. The fourth patient showed the upper esophagus to be somewhat dilated. The walls were flabby and in the middle third were marked with longitudinal, streaky esophagitis.

It is reasonable to suppose that the endoscopic findings will vary a great deal with the stages at which the examinations are made. Watson-Williams described the characteristic findings at esophagoscopy as "general esophagitis tending to affect the middle two-fourths of the esophagus; maceration of the epithelium with longitudinal desquamation going on to complete shedding of the epithelium; formation of leukoplakic patches consisting of thickened epithelium only." The exact locations of the lesions in the various reports are not always given. However, the "upper end" of the esophagus was affected twice as often as the "lower end," six lesions being noted in the upper part and three in the lower part. In all of Watson-Williams' cases the middle two-fourths was given as the site of affection.

DISCUSSION

The diagnosis of tertiary syphilis of the esophagus will not often be made. In spite of this, it seems reasonable that in view of the prevalence of syphilis and the possibility that syphilitic dysphagia may occur much more commonly than is generally accepted, the condition should be thought of in the case of every patient complaining of dysphagia.

The blood serology may or may not be positive and is only of leading diagnostic significance, since an unknown number of those suffering any form of obstruction of the esophagus will have positive Wassermann and Kahn tests.

Positive biopsy findings will establish some of the diagnoses. It should be borne in mind that biopsy in the esophagus is frequently very difficult. Avery[11] felt that a history of transitory attacks of mild dysphagia is very suggestive of syphilis of the esophagus.

Guyot[1] considered as authentic cases of syphilis of the esophagus only those (a) recognized at post-mortem and (b) those found at esophagoscopy and cured by specific treatment. The diagnosis of tertiary syphilis of the esophagus should not be made on the basis alone of relief of dysphagia by specific treatment, because dysphagia may be due to gumma in the mediastinum, located in such a position as to cause partial obstruction by pressure, while not primarily involving the esophagus.

The facts that the clinical course has been shown to be prolonged in many of the patients, that there is generally absence of pain, and that the lesion occurs most frequently in the upper end of the esophagus should serve as guides to the diagnosis. Thus, in a patient who has had syphilis and who gives a history of long-standing dysphagia without pain, tertiary syphilis of the esophagus should be considered. If an intrinsic obstruction can be shown by x-ray or by esophagoscopy, or both, the diagnosis of tertiary syphilis of the esophagus becomes probable. If such a patient is cured by specific antisyphilitic treatment, the diagnosis is then established. It should be remembered in this connection that a number of patients will not respond favorably to specific treatment—those who have extensive scarring and resultant fibrotic stenoses.

While secondary syphilis of the esophagus has not been reported, Wile[17] offered the interesting speculation that the dysphagia occasionally occurring in secondary syphilis may be due to involvement of the esophagus by secondary lesions.

TREATMENT

The treatment is that of tertiary syphilis. In fairly early cases or in cases where extensive scarring and stenosis have not occurred, it appears that cure will be prompt on drug treatment alone. Where the esophagus has been severely scarred and a fibrotic obstruction has occurred, dilatation will be required. When the stenosis is undilatable and is unrelieved by drug treatment, esophagectomy with esophagogastrostomy may be considered.

When a patient with tertiary syphilis of the esophagus is known to have a tracheoesophageal fistula, it is important to keep him flat on his back at all times. This was pointed out by Lukens and Ono[5] whose patient had such a fistula and who coughed severely on swallowing liquids in all positions except the supine. If the outlook in patients with tracheoesophageal fistulas is otherwise good, tube feeding should be done. If this is impossible for any reason, temporary gastrostomy should be done. This is justified because the patient will stand a good chance of being cured if aspiration pneumonitis can be prevented.

SUMMARY

1. A case of tertiary syphilis of the esophagus is presented.

2. The literature is reviewed.

3. A history of long-standing dysphagia of a painless nature, with positive serology and x-ray or endoscopic evidence of intrinsic esophageal obstruction, is thought to be suggestive of the presence of tertiary syphilis of the esophagus.

4. The treatment is primarily medical but dilatations of the esophagus are indicated where stenoses are present.

5. The prognosis is favorable except in patients with tracheoesophageal fistulas. In these the prognosis appears favorable if aspiration pneumonitis can be prevented.

REFERENCES

1. Guyot, Raymond: Syphilis of the Esophagus: Report of Two Cases, Ann. d'oto-laryng. pp. 505-526, May, 1931.
2. Moore, Alexander B.: Benign Lesions in Cardiac Portions of the Esophagus and Stomach: Roentgenologic Diagnosis, J. A. M. A. 24: 12-15, 1930.

3. Guisez, J.: Syphilis of the Esophagus, Bull. de med. **90**: 483-486, 1924.
4. Vezina, Charles: A Case of Syphilis of the Esophagus, Laval méd. **1**: 279-281, 1936.
5. Lukens, R. M., and Ono, J.: Syphilitic Tracheoesophageal Fistula: Report of Case, Laryngoscope **44**: 334-339, 1934.
6. Shimokawa, H.: Tracheoesophageal Fistula Due to Acquired Syphilis: Case, Taiwan Igakkai Zassi (Abstr.) **32**: 165-166, 1933.
7. Fontana, Dino: Syphilitic Stenosis of the Esophagus With Symptoms Simulating an Esophagotracheal Fistula, Ann. di laring otol. **38**: 23-29, 1938.
8. Hanif, I.: Case of Syphilis of the Esophagus, Türk tib cem. mec. **1**: 144-145, 1935.
9. Wilcox, Leslie F.: Tertiary Syphilis of the Esophagus With Report of Case Recognized Roentgenologically, Am. J. Roentgenol. **31**: 773-777, 1934.
10. Glass, W. E., and Freeman, W.: Spontaneous Rupture of the Esophagus in Syphilis, Am. J. Med. Sc. **189**: 80-86, 1935.
11. Avery, Philip: Syphilis of the Esophagus, Radiology **27**: 323-327, 1936.
12. Myerson, C.: Syphilis of the Esophagus, M. Clin. North America **10**: 919-925, 1927.
13. Ponzoni, V.: Syphilis of the Esophagus: Case, Policlinico (sez prat.) **40**: 1209-1212, 1933.
14. Watson-Williams, E.: Syphilis of the Oesophagus, Practitioner **131**: 310-312, 1933.
15. Sabalette, P. R., and Martinez, E. G.: Esophageal Gumma Perforated Into Trachea: A Case, Med. clin. Barcelona **4**: 105-107, 1945.
16. Postigo, J. C.: Syphilis of Esophagus: A Case, Rev. clin. españ. **8**: 199-200, 1945.
17. Wile, Udo J.: Syphilis of the Esophagus, Am. J. Med. Sc. **148**: 180-186, 1914.

CAVERNOSTOMY FOR RESIDUAL CAVITY AFTER THORACOPLASTY

Early Closure of Cavernostomy Sinuses Under Streptomycin Protection

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EXTRAPLEURAL thoracoplasty continues as the most commonly used major surgical procedure in the treatment of pulmonary tuberculosis. The results of thoracoplasty in terms of cavity closure vary widely in reports of different surgeons. Dolley, Jones, and Paxton[1] reported a series of 1,636 thoracoplasty operations in 1939. Among these patients 24.2 per cent were considered failures due to residual cavity and active endobronchial disease. O'Brien, Day, Chapman, and Tuttle[2] reported residual cavities in 12.98 per cent of 511 thoracoplasty patients. O'Brien[3] in objecting to routine preoperative needling of cavities, stated:

> "We have well over 1,000 thoracoplasty patients and our percentage of cavity closure will run from 80 to 90 per cent. Almost any report on a group of thoracoplasties will be the same."

Krynski[4] reported a series of 308 cases of thoracoplasty from Sea View Hospital in 1947, in which 51.3 per cent were considered arrested. The operation has become rather well standardized by several generations of thoracic surgeons and nearly all thoracoplasties now done are technically good operations. The cause of the variation in number of residual cavities reported by different authors probably lies in the selection of cases rather than in the technique of the operation. The deeper one goes in the barrel in the effort to salvage some of these questionable risks the higher will be the number of residual cavities.

At the Veterans Administration Hospital, Oteen, N. C., 525 patients have undergone a thoracoplasty in the past five years. In 20 per cent of these a residual cavity has persisted. There have been minor variations in technique in that an anterior stage was occasionally used if the cavity was in an anterior position and the processes removed or the heads of the ribs disarticulated if the cavity was proved to be in the posteromedial portion of the upper lobe. These variations have not been considered determinant factors in the number of residual cavities encountered.

The success attained in eradicating these residual cavities often determines the success of the entire surgical program. It is satisfying to obtain a negative sputum after a thoracoplasty, but it is doubly so, both to the surgeon and the patient, to attain success after an initial failure. The methods used and the results obtained in dealing with these cavities at Oteen have been described in

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another paper.[5] The insertion of a Monaldi catheter concomitantly with the thoracoplasty, as described by Vineberg and Kunstler,[6] has been used in a number of patients without alteration in the final results.

At the present time, when confronted with a postthoracoplasty cavity, resection is given first consideration. Lobectomy or pneumonectomy for residual cavity[7, 8] has been done on 44 patients at Oteen since January, 1947. The total mortality rate following these resections has been 6.8 per cent. Ninety-one and two-tenths per cent of the living patients now have a negative sputum. Wound infection, empyema, and bronchopleural fistula complicate such operations frequently enough to act as a deterrant to unbridled enthusiasm for the method.

Some patients, because of age, diminished pulmonary function, contralateral disease, or the size and location of the cavity, are not considered suitable risks for resection. In this group of patients we are now using open cavernostomy to obliterate the cavities. The discovery of streptomycin, in 1944, by Schatz, Bugie, and Waksman[9] and the report of its effectiveness against the tubercle bacillus in December of the same year by Feldman and Hinshaw[10, 11, 12] caused us to use this drug as an adjunct to permit early closure of the resultant cavernostomy sinuses.

The history of cavernostomy, the surgical technique employed, and the results obtained in a group of 22 patients followed for periods of four to twenty-three months are presented in this paper. Attention is given to the use of streptomycin as an adjunct to the procedure.

HISTORY

Cavernostomy, speleotomy, or open drainage of tuberculous cavities was probably the earliest form of surgical treatment used for pulmonary tuberculosis[13, 14] and may well date back to the time of Hippocrates.[15, 16] The earliest reported case of incision of a tuberculous cavity is that of Pheraeus who received a spear thrust in battle which opened a cavity in his lung and restored his health.[17] In the famous de Bligny case, the son of M. de la Genevraye, a victim of phthisis had abandoned all hope of recovery, in the year 1670, when he received a sword wound which opened a cavity in the right lung and allegedly cured his tuberculosis.[17, 18, 19]

Godlee[20] stated that Paschal, Paré, and Harvey incised the chest in desperate cases and drained tuberculous cavities. Baglivi,[21] in 1696, believed that opening a cavity led to cicatrization and advised an intercostal incision to permit the introduction of medicaments into the cavity.

Le Carboulec[19] recorded the opinions of Barry, Boerhaave, Sharpe, Pouteau, David, and Bell who opened tuberculous cavities with a lancet or trocar. According to Elliott,[22] as related by Hastings,[23] a Dr. E. Barry of Dublin,[24] in 1727, recommended puncturing tuberculous cavities as a means of curing phthisis and, in 1763, reported several cases in which he had successfully used this method. Sharpe[25] of Ireland, in 1769, recommended incising tuberculous cavities if pleural fusion had occurred and introduced a gauze drain. Bell[26] of Scotland, in 1778, referred to free incision of "abscesses" in the lung and drainage by means of lead pipes. Gumprecht[27] of Göttingen, in 1793, also advocated incision of tuberculous cavities.

In the nineteenth century, as stated by Le Carboulec,[19] incision and drainage of tuberculous cavities was attempted or performed by Ramadge (1814), Calissen (1815), Zang (1818), Nasse (1827), Krimer (1829), and Hoekten (1844). About 1830, March (Dublin), according to Hastings,[22, 23] incised and drained tuberculous cavities in two patients, one of whom recovered. Hastings, however, believed the diagnosis in the successful case to be a pulmonary abscess or empyema necessitatis. About the same time Thomas of Wales performed a similar operation. In 1845, Hastings[23] reported two patients with apical pulmonary cavities in whom he procured the help of Stokes, a surgeon, to open and drain the cavities by insertion of a rubber tube. Improvement occurred in one case.

Mosler,[28] in 1873, punctured a tuberculous cavity with a trocar and cannula, widened the wound with forceps, packed it with gauze, and inserted a large silver drainage tube through which he irrigated the cavity with a dilute solution of potassium permanganate. Thereafter cavernostomy was performed with increasing frequency. Prior to 1885, incision and drainage of tuberculous cavities had been carried out by incision, or insertion of a trocar and cannula, through an intercostal space without resection of any portion of the thoracic cage. In the development of cavernostomy and in the advancement of thoracic surgery much credit is due de Cérenville of Lausanne, Switzerland, because he not only performed the first thoracoplasty for pulmonary tuberculosis, but combined this procedure with cavernostomy. In 1885,[29] he reported four cases of pulmonary apical cavities in which he resected portions of the second and third ribs anteriorly and in two opened the cavities intentionally. For postoperative dressings he used gauze, cotton, iodoform tampons, alum, thymol, balsam, and caustic agents. Improvement, however, was only temporary.

In 1891, Sonnenburg[30] reported his famous case of cavernostomy in which, under Koch's direction, he resected the first rib and cartilage, opened into the cavity with thermocautery, and injected tuberculin into the wound which was packed with gauze. The result was remarkable and the patient was well five years after operation. Tuffier,[31] in 1944, collected 45 cases of cavernostomy. Seventeen of these terminated fatally within three months after operation. He concluded that Sonnenburg's case was the only one with an incontestable favorable result.

In the second decade of the twentieth century Sauerbruch of Germany contributed greatly to the development of cavernostomy. In 1920,[32] he presented the theory that a reduction in the size of the thoracic cage should be obtained prior to cavernostomy, either by a preliminary thoracoplasty or by extrapleural pneumonolysis or both. In his publication in 1930,[14] he described the technique of resection of portions of the overlying ribs. The cavity was then entered by incision or cautery and packed with gauze. He also stated that after two or three months a soft tissue plastic operation could be performed to close the fistula.

Cavernostomy was of interest to European surgeons for many years prior to its introduction in the United States. It was not until 1924 that Gekler, Rankin, and Weigel[33] reported three cases of cavernostomy in which post-

operatively gentian violet and methylene blue were instilled into the cavities to produce chemical sterilization, with favorable results. Lilienthal, in 1927[34] and 1929,[35] reported a total of four cases of cavernostomy for residual cavity after thoracoplasty and in 1931,[36] reported two additional cases in which he inserted a pedicled muscle flap into the cavity, thus applying the original method devised by Pool and Garlock[37] for closure of persistent bronchial fistula. He reported success in one instance and failure in the other. Bruns and Casper,[38] in 1932, reported 22 cases of cavernostomy with disappointing results. In 1935, Eloesser[39] devised his U-shaped skin flap technique for drainage of tuberculous empyema and shortly thereafter he and others adapted this technique to cavernostomy. In 1937, Neuhof[40] advocated closure of residual bronchocutaneous fistula after cavernostomy by means of a fat transplant when simple plastic repair failed. Alexander,[41] in 1937, mentioned the use of a pedicled muscle graft to close residual bronchocutaneous fistula.

Up to comparatively recent years cavernostomy has been fraught with so many dangers, difficulties, and poor results that none of the many surgeons who have attempted it have continued to use it as a standard procedure. Revival of interest in cavernostomy came along with new concepts or theories as to the mechanics and aerodynamics of formation and closure of cavities. Hall,[42] in the British Isles in 1922, after study of pathologic specimens, first called attention to the importance of the condition of the bronchus draining a cavity and described a flap valve mechanism. Ameuille and Levesque (1923),[43] Eloesser (1934),[44] and Korol (1935)[45] also emphasized the importance of the state of the connecting bronchus. Coryllos,[46] in 1936, made a meritorious contribution when he postulated that changes in the size of cavities and their closure or resistance to healing were due to the state of the connecting bronchial openings. He and Ornstein[47] acted on the theory that the cavity would close if the draining bronchus was obliterated. They attempted this obliteration by (1) an intracavitary transthoracic injection into the cavity of various substances, (2) cauterization of the bronchial openings through a cavernoscope, and (3) intracavitary transplantation of a pedicled muscle flap. The report of their results in 1938 was not encouraging.

Eloesser,[48, 49] Salkin, Cadden, and McIndoe,[50] Shipman,[51] Pinner,[52] Auerbach and Green,[53] Brunn, Shipman, Goldman, and Ackerman,[54] Goldman,[55] Shipman, Rogers, and Daniels,[56, 57] and others recognized the important role of the draining bronchus in "tension cavities," but disagreed as to the necessity of closing the draining bronchus to bring about closure of the cavity. They recognized the fact that other factors, termed by Eloesser as extrinsic, also played a role in the formation and closure of cavities. Other authors also contributed to our modern understanding of cavity mechanics. Among these are, Pearson,[58] Brooke,[59] Ameuille, Delhomme, and Raust,[60] Loesch,[61] Thomas,[62] Meyersburg, Gruber, and Lupo,[63] Riggins and Gearhart,[64] Maier,[65] LeCarboulec,[19] Vineberg and Kunstler,[6] Bernou,[66] Bobrowitz,[67] Brock,[68] and Even and Lecoeur.[69]

Among the most detailed studies of cavernostomies in recent years have been those of Le Carboulec,[19, 70] Hochberg, Fink, and Chardack[71] and O'Brien, O'Rourke, Test, and Skinner.[72] Le Carboulec used the operation only for

residual postthoracoplasty cavities and emphasized the necessity of weekly postoperative cauterization of the walls of the cavity and the bronchial orifices. He stated that contrary to some other authors he had always found several bronchial openings communicating with the cavity. The purpose of the cautery in his opinion is not to obliterate the bronchial orifices and achieve cure by atelectasis but to destroy the tuberculous focus at its site. By relieving the cavity of its caseous burden it then is permitted to heal by swelling and contraction of its walls. In his series of 21 cases there were two operative deaths. Among the 19 survivors, 13 had a negative sputum and a healed wound. In 6 patients a sinus persisted or there was an ipsi- or contralateral exacerbation of disease. The average duration of drainage in the healed cases was 12 months. The author stressed the fact that cavernostomy should be used only in postthoracoplasty cavities in whom the cavity represents the sole remaining focus of disease.

In 1945, O'Brien and associates reported a group of 74 patients upon whom they had performed a cavernostomy. Among these were 13 patients who had a residual postthoracoplasty cavity. Fifty-five per cent of the 11 patients who survived in this group were reported as healed and arrested. Nine per cent were improved, 9 per cent unclassified and 27 per cent dead. Among the living patients of the entire series who had been operated on earlier than one year prior to the preparation of the report wound healing occurred in 56 per cent. These authors did not use the skin flap technique.

TECHNIQUE

The operation is performed in two or more stages. The cavity is first localized with posteroanterior and lateral x-ray films. A lead marker is fixed to the skin in the subclavicular and midaxillary area before exposure to aid in localization. Planigrams are also taken to determine the level of the cavity and for comparison in the postoperative determination of cavity closure. In most cases the best site for approach to the cavity has been found to be the midaxillary region.

FIRST STAGE

The first stage is performed under general anesthesia. A U-shaped flap of skin is raised with the free end distally. The flap is made long enough to permit insertion of the free end into the cavity when it is opened at a later date. Excess fatty tissue is removed. Regenerated ribs and muscle bundles overlying the cavity are excised over a wide area. The parietal pleura is exposed by dissection with small "bronchial sponges" and as large an extrapleural space as possible developed. The free end of the skin flap is then tucked into the wound which is packed with plain gauze. A metal skin clip is fixed to the end of the gauze packing and placed at the projected site of the cavernostomy. This serves as a guide to the best approach to the cavity when later x-ray studies are made. The wound is then closed as tightly as possible around the skin flap with interrupted chromic No. 1 sutures for the deep tissues and nylon sutures for the skin.

SECOND STAGE

The second stage is performed 10 to 14 days later. There is occasionally a moderate amount of seropurulent drainage from the wound during this interval. Local anesthesia is used for the second stage in order to preserve the cough reflex and aid the patient to expel any blood or pus that might enter the bronchial tree. The wound is reopened and the pack removed. The site of the skin clip is noted and compared with x-ray films to aid in localizing the cavity. Pleural fusion is first assured by needle aspiration. An attempt is then made to enter the cavity with an 18-gauge needle attached to a syringe. It is wise to sound a note of caution at this point. Following a thoracoplasty the superior portion of the upper lobe is compressed to a narrow tongue of tissue which extends upward between the mediastinum and the collapsed chest wall. On the left side the aorta must be avoided as well as the subclavian vessels. On the right side the azygos vein and the superior vena cava are endangered. When the cavity is entered, manometric readings are taken before and after injection of 10 c.c. of air through the needle and following withdrawal of 20 c.c. of air. These readings serve as an indicator as to whether or not we are dealing with a blocked cavity. If the bronchus draining the cavity is open the pressure readings will not be altered by introduction or withdrawal of air. If the cavity is blocked by a valve the readings will be elevated to +8 to +14 on introduction of 10 c.c. of air. When 20 c.c. of air is then withdrawn readings of –3 to +3 are obtained. As large an area of the cavity as possible is then unroofed. This may be done by the electrocautery blade or, if the large vessels are threatened, by sharp dissection. The distal end of the skin flap is then sutured to the cavity wall with mattress sutures of chromic No. 1 gut and a gauze pack placed into the cavity and wound to hold the flap in place. If the site is too inaccessible the sutures are omitted and the gauze pack relied upon to hold the flap in situ. We have often failed to locate the cavity at this stage and have then repacked the wound and made further x-ray studies before finally entering the cavity.

POSTOPERATIVE CARE

If the cavity is successfully entered the pack is left in place for four days. It is then removed and the sinus packed daily with gauze moistened with an antiseptic solution. Formerly we used one composed of equal parts of penicillin (250 units per c.c.) and a 1 per cent solution of acetic acid. At present we are using Chloresium. This regime has kept the wound clean and free of pyocyaneus infection which we occasionally encountered prior to its use. Penicillin is administered intramuscularly for a few days before and after each stage until the temperature reaches the normal level. It is necessary to gently dilate the cavity stoma with the gloved finger at weekly intervals in order to prevent too rapid contraction of the opening.

CLOSURE OF THE CAVERNOSTOMY

After a period varying from three to five months the drainage usually becomes thin and scanty. The sinus tract narrows and epithelializes. The

TABLE I

CASE NO.	CAVITY		STREPTOMYCIN		RESULTS			COMPLICATIONS	MONTHS SINCE CLOSURE
	OPENED	CLOSED	BEGAN	ENDED	CAVITY	SINUS	SPUTUM		
F. B. 1	1-26-46	3-14-47	2-28-47	3-28-47	Equivocal	Closed	Neg.	None	24
F. R. 2	8-21-46	—	—	—	—	—	—	Traumatic pneumothorax, contralateral spread, died 26 days postop.	—
Z. S. 3	11-25-46	5-13-47	4-12-47	8- 7-47	Closed	Closed	Neg.	None	22
J. W. 4	12-13-46	4- 9-47	3-25-47 4-30-47	4-23-47 5-19-47	Closed	Closed	Neg.	None	23
E. J. 5	1-22-47	5-13-47	4-11-47	6-13-47	Closed	Closed	Neg.	Traumatic pneumothorax, 2nd stage, re-expanded	22
C. P. 6	2-24-47	5-15-47	4-17-47	6-12-47	Closed	Closed	Neg.	None	22
S. R. 7	2-24-47	6-20-47	5-24-47	8-10-47	Closed	Closed	Neg.	None	21
J. B. 8	3-12-47	7-25-47	6-25-47	9-22-47	Open	Open	Pos.	Primary healing, late recurrence	13
E. B. 9	5- 5-47	8-22-47	7-21-47	10-10-47	Closed	Closed	Pos.	Late contralateral cavitation	19
D. L. 10	5- 5-47	10- 8-47	9- 2-47	11-20-47	Closed	Closed	Neg.	Traumatic pneumothorax, 2nd stage, re-expanded	17
J. P. 11	6- 4-47	9-18-47	8-14-47	10-24-47	Closed	Closed	Neg.	None	18
C. J. 12	8-28-47	3- 3-48	2- 4-48	4- 5-48	Closed	Closed	Neg.	None	13
W. P. 13	9-12-47	4- 2-48	3- 2-48	5- 4-48	Closed	Closed	Neg.	None	12
T. P. 14	10-15-47	4- 5-48	3- 2-48	5- 4-48	Closed	Closed	Neg.	None	12
O. P. 15	10-24-47	—	—	—	Open	Open	Pos.	Contralateral reactivation	—

R. L. 16	12- 5-47	4- 6-48 7-12-48 9- 3-48	4- 7-48	8- 3-48	Closed	Intermittent drainage	Neg.	Persistent sinus	7
A. Y. 17	4- 6-48	11- 1-48	10-28-48	11- 7-48	Closed	Closed	Neg.	None	6
T. T. 18	4- 6-48	—	—	—	Open	Open	Pos.	Ipsilateral basal cavitation	—
W. L. 19	5-12-48	10-29-48	10-27-48	11-12-48	Closed	Closed	Neg.	None	5
W. S. 20	9-27-48	—	—	—	Open	Open	Pos.	Late hemoptysis	—
K. S. 21	11- 5-48	—	—	—	Open	Open	Neg.	Ipsilateral pleural effusion, late	—
J. G. 22	12- 9-48	—	—	—	Open	Open	Pos.	Traumatic pneumothorax, 1st stage, re-expanded	—
W. P. 23	2-21-49	—	—	—	Open	Open	Neg.	None	—

sputum is studied by concentration and culture and the sinus secretions by culture for acid-fast organisms. If three successive cultures of both the sputum and drainage material are negative and planigrams indicate an eradication of the cavity, closure is attempted. Our present policy is to administer a 1 Gm. daily dose of streptomycin for one week preoperatively and two weeks postoperatively. This is divided into two doses of 0.5 Gm. each.

Under general inhalation anesthesia given through an intratracheal catheter, the skin flap is freed and the sinus tract excised. As much regenerated bone as possible is again removed from around the sinus and a pedicle graft developed from the edge of the pectoralis minor muscle. The free end of the graft is fixed in place at the site of the previous cavity by mattress sutures of No. 1 chromic gut. The excess skin flap is excised and the wound closed with interrupted chromic No. 1 sutures for the deeper structures and nylon for the skin.

COMPLICATIONS

Traumatic pneumothorax occurred during four operations. One patient died, the others re-expanded, and the operation was later completed. Reactivation of disease occurred in 3 patients. One was the patient who died. He had a moderate contralateral reactivation at the time of death. Another patient developed a second cavity in the lower lobe some distance posterior to the original cavity which had been drained anteriorly. The posterior cavity was opened from a posterior approach. The third is clinically improved although the sinus has not been closed.

RESULTS

The results of the entire series are given in Table I.

The combined results are given in Table II.

TABLE II. RESULTS OF CAVERNOSTOMY SERIES

CAVITIES OPENED	CAVITIES CLOSED	DEATHS		SPREADS		NEGATIVE SPUTUM IN CLOSED SINUSES		RECURRENCE IN CLOSED SINUSES	
		EARLY	LATE	NUMBER	PER CENT	NUMBER	PER CENT	NUMBER	PER CENT
23	16	1	0	2	8.7	13	81.2	2	12.5

Twenty-three patients with residual postthoracoplasty cavities have had their cavities drained by an open cavernostomy. Sixteen have had the resultant sinus tract closed by a muscle plastic operation, using streptomycin as an adjunct. Thirteen of these have been closed and followed for periods varying from one to two years.

One patient died following the first stage operation. Traumatic pneumothorax was incurred at the operation and a contralateral spread followed. Death occurred suddenly on the twenty-first postoperative day, apparently of a pulmonary embolism.

Seven patients still have a draining sinus. Three of these are recent cases and will ultimately be closed. The remaining three were poorly selected cases

and cannot be closed. The prerequisite established by Le Carboulec that the cavity must represent the sole remaining focus of disease was not followed. One of these patients had caseous disease in the entire lung and although the cavity has apparently been eradicated a pneumonectomy will ultimately be required. One patient has required a second drainage of another cavity in the same lung. He has shown dramatic clinical improvement and recently had a negative concentrated sputum for the first time in five years. The third patient represented a salvage attempt. A giant cavity occupied an entire lung in a patient with extreme toxic symptoms. A Monaldi drainage was followed by a thoracoplasty and finally an open cavernostomy. He also has shown dramatic clinical improvement. Contralateral cavitation is being controlled. No effort has been made to close the sinus.

Sixteen patients had the sinus closed by a muscle plastic operation. In three patients, tissue sections of the excised skin flap were made at the time of closure. The findings of Coryllos[47] were confirmed in these for typical tubercles were found in the section. All wounds except one healed by primary intention or after one to two weeks of drainage. In one patient, despite three attempts at closure a small sinus continues to drain intermittently. One patient whose wound had healed by primary intention had a recurrence of the sinus and a positive sputum thirteen months later. A bilobectomy was then done and the patient now has a healed wound and a negative sputum.

Thirteen of the patients whose sinuses were closed have a negative sputum. The patient with the persistent sinus has an occasional positive sputum. The patient with the persistently positive sputum obtained a successful result following the cavernostomy. He had a negative sputum for several months following closure. He is an alcoholic, however, and ultimately developed a contralateral cavity. The patient who had a lobectomy for recurrent cavity and positive sputum now has a negative sputum.

The residual cavity has been definitely eradicated, as revealed by planigraphic x-ray studies in 12 of the 16 patients whose sinuses were closed. Three of the remainder have planigraphic x-ray studies consistent with bronchiectasis in the former cavitary area. Two of these have a consistently negative sputum nearly two years after closure. One definite recurrence of sinus and cavity required lobectomy.

CONCLUSIONS

1. The technique of cavernostomy in a consecutive series of 22 patients who had a residual cavity after thoracoplasty has been presented.

2. The use of streptomycin as a protective agent in surgical plastic closure of cavernostomy openings in 16 patients has been discussed. Streptomycin has made possible the early closure of sinuses resulting from cavernostomies with complete healing of 93.7 per cent of 16 attempts.

3. Cavernostomy with streptomycin as an adjunct offers a satisfactory form of treatment for patients with residual cavities who are not suitable risks for resection.

REFERENCES

1. Dolley, F. S., Jones, J. C., and Paxton, J. R.: Late Results of Thoracoplasty, Evaluation of Results From 14 American Clinics in 1,636 Cases, Am. Rev. Tuberc. **39**: 145-161, 1939.
2. O'Brien, E. J., Day, J. C., Chapman, P. J., and Tuttle, W. M.: A Study of the Immediate and Late Results in 511 Patients Subjected to Thoracoplasty, J. THORACIC SURG. **9**: 364-381, 1940.
3. O'Brien, E. J.: In discussion of article by Head, J. R.: Intracavitary (Monaldi) Suction, J. THORACIC SURG. **15**: 156-157, 1946.
4. Krynski, B.: Survey of Thoracoplasties at Sea View Hospital Over a Five-Year Period (1937-1942), Quart. Bull. Sea View Hosp. **9**: 144-154, 1947.
5. Murphy, J. D., Elrod, P. D., Walkup, H. E., and Koontz, E. R.: Surgical Treatment of Residual Cavities Following Thoracoplasties for Tuberculosis, Dis. Chest **14**: 694, 1948.
6. Vineberg, A. M., and Kunstler, W. E.: The Determination and Treatment of Pressure Cavities in Pulmonary Tuberculosis, Surg., Gynec. & Obst. **78**: 245-274, 1944.
7. Murphy, J. D., Moore, J. A., and Elrod, P. D.: An Evaluation of Streptomycin as a Protective Agent in Pulmonary Resections for Tuberculosis, J. THORACIC SURG. **18**: 45, 1949.
8. Moore, J. A., Murphy, J. D., and Elrod, P. D.: The Use of Streptomycin in Pulmonary Resections, S. Clin. North America (Dec.) 1948.
9. Schatz, A., Bugie, E., and Waksman, S. A.: Streptomycin, Substance Exhibiting Antibiotic Activity Against Gram-positive and Gram-negative Bacteria, Proc. Soc. Exper. Biol. & Med. **55**: 66-69, 1944.
10. Feldman, W. H., and Hinshaw, H. C.: Effects of Streptomycin on Experimental Tuberculosis in Guinea Pigs; a Preliminary Report, Proc. Staff Meet., Mayo Clin. **19**: 593-599, 1944.
11. Hinshaw, H. C., and Feldman, W. H.: Streptomycin in Treatment of Clinical Tuberculosis; a Preliminary Report, Proc. Staff Meet., Mayo Clin. **20**: 313-318, 1945.
12. Hinshaw, H. C., Feldman, W. H., and Pfuetze, K. H.: Treatment of Tuberculosis With Streptomycin: Summary of Observations on 100 Cases, J. A. M. A. **132**: 778-782, 1946.
13. Eloesser, L., Rogers, W. L., and Shipman, S. J.: Treatment of Insufflated Cavities, Am. Rev. Tuberc. **51**: 7-17, 1945.
14. Sauerbruch, F.: Die Chirurgie der Brustorgane, ed. 3, vol. 2, Berlin, 1930, Julius Springer, p. 1096.
15. Krause, A. K.: A Note on the Practice of Artificial Pneumothorax by the Hippocratic School, Am. Rev. Tuberc. **6**: 327-330, 1922. Reference to and partial translation of Oeuvres complètes d'Hippocrate, Vol. 7, Book 2, sect. 59, E. Littré, Paris, 1851, J. B. Baillière.
16. Davidson, L. R.: Cavernostomy in Pulmonary Tuberculosis, Clinics **3**: 1179-1186, 1945.
17. Paget, S.: Surgery of the Chest, Bristol, John Wright & Co.; London, 1896, Simpkin, Marshall, Hamilton, Kent & Co., Ltd., chap. 21, pp. 330-335.
18. Brewer, G. E.: Surgery of the Thorax, in Surgery; Its Principles and Practice, Vol. 3, edited by Keen, W. W., Philadelphia, 1916, W. B. Saunders Company, pp. 544-545.
19. Le Carboulec, G.: La Spéléotomie, Niort, 1945, Imprimerie St. Denis, pp. 1-187.
20. Godlee, R. J.: Lectures on the Surgical Treatment of Pulmonary Cavities, Lancet **1**: 457-459, 1887; **1**: 511-513, 1887.
21. Baglivi: De praxi medica ad priscam observandi rationem revocanda, Romae **2**: 249, 1696. Referred to by Vineberg and Kunstler[6] and by Le Carboulec.[19]
22. Elliott, J. H.: Hastings on Catheter Drainage of Tuberculous Cavities, Am. Rev. Tuberc. **46**: 546-548, 1942.
23. Hastings, J.: Pulmonary Consumption Successfully Treated With Naphtha and an Appendix Showing the Utility of Puncturing Tuberculous Cavities as an Adjunct in the Cure of Phthisis, ed. 2, London, 1845, John Churchill, quoted by Elliott.[22]
24. Barry, E.: A Treatise on a Consumption of the Lungs, Dublin, 1726, p. 217.
    Idem: A Treatise on Three Different Digestions, London, 1763, p. 366. Quoted by Elliott[22] and Hastings.[23]
25. Sharpe, E.: A Treatise on the Operations of Surgery, ed. 9, London, 1769, p. 128. Quoted by Vineberg and Kunstler[6] and by Le Carboulec.[19]
26. Bell, B.: A System of Surgery, ed. 3, London, 1799, Vol. 5, chap. 36, sect. 11. Quoted by Godlee.[20]
27. Gumprecht: De pulmonum abscesses ope chirurgia aperiendo, Göttingen, 1793. Quoted by Godlee.[20]
28. Mosler, F.: Separatabdruck, Klin. Wchnschr. No. 43, 1873. Quoted by Godlee.[20]
29. De Cérenville, E. B.: De l'intervention opératoire dans les maladies du poumon, Rev. méd. de la Suisse Rom. **5**: 441-467, 1885.

30. Sonnenburg: Die chirurgische Behandlung der Lungenkavernen, Deutsche med. Wchnschr. **1:** 21, 1891. Quoted by Le Carboulec.[19]
31. Tuffier, T.: Etat actuel de la chirurgie intrathoracique, Paris, 1914, Masson & Cie, pp. 90-105; 163-172. Quoted by Le Carboulec.[19]
32. Sauerbruch, F.: Die Chirurgie der Brustorgane, ed. 2, Vol. 1, Berlin, 1900, Julius Springer.
33. Gekler, W. A., Rankin, H. P., and Weigel, B. J.: Tuberculous Cavitation of the Lung; Mechanical Factors in Its Genesis, and Combined Chemotherapeutic and Surgical Treatment, J. A. M. A. **82:** 457-463, 1924.
34. Lilienthal, H.: Mechanical Principles of the Operative Treatment of Pulmonary Tuberculosis, Ann. Surg. **86:** 182-199, 1927.
35. Lilienthal, H.: Direct Drainage of Tuberculous Pulmonary Cavities, Arch. Surg. **19:** 1161-1168, 1929.
36. Lilienthal, H.: Operative Treatment of Phthisis; the Surgeon's Moral Obligation, Am. J. Surg. **14:** 356-381, 1931.
37. Pool, E. H., and Garlock, J. H.: Treatment of Persistent Bronchial Fistula; an Experimental and Clinical Study, Ann. Surg. **90:** 213-237, 1929.
38. Bruns, E. H., and Casper, J.: The Present Status of Chest Surgery in the Treatment of Pulmonary Tuberculosis, With Special Reference to Thoracoplasty, Am. Rev. Tuberc. **26:** 665-688, 1932.
39. Eloesser, L.: An Operation for Tuberculous Empyema, Surg., Gynec. & Obst. **60:** 1096-1097, 1935.
40. Neuhof, H.: The Free Transplantation of Fat for the Closure of Bronchopulmonary Cavities (Lattice Lung), J. THORACIC SURG. **7:** 23-37, 1937.
41. Alexander, J.: The Collapse Therapy of Pulmonary Tuberculosis, Springfield, Ill., 1937, Charles C Thomas, ch. 29, pp. 580-586.
42. Hall, R.: Some Rare and Obscure Pulmonary and Pleural Conditions Lancet **1:** 61-63, 1922.
43. Ameuille, P., and Levesque: La Broncheo de Drainage des Cavernes Tuberculeuses, Bull. et mem, soc. med. d' hop. de Paris **47:** 612-614, 1923. Quoted by Vineberg and Kunstler.[6]
44. Eloesser, L.: Bronchial Stenosis in Pulmonary Tuberculosis, Am. Rev. Tuberc. **30:** 123-180, 1934.
45. Korol, E.: The Alternately Expanding and Contracting Tuberculous Cavity, Am. Rev. Tuberc. **31:** 475-479, 1935.
46. Coryllos, P. N.: The Mechanics and Biology of Tuberculous Cavities, Am. Rev. Tuberc. **33:** 639-660, 1936.
47. Coryllos, P. N., and Ornstein, G. G.: Giant Tuberculous Cavities of the Lung—Pathogenesis, Pathologic Physiology, and Surgical Treatment, J. THORACIC SURG. **8:** 10-54, 1938.
48. Eloesser, L.: Blocked Cavities in Pulmonary Tuberculosis, J. THORACIC SURG. **7:** 1-22, 1937.
49. Eloesser, L.: The Choice of Procedure in the Treatment of Tuberculous Cavities; Considerations Regarding Intracavitary Pressure Based on Clinical Observations and Experiments on Cadavers, J. THORACIC SURG. **10:** 501-528, 1941.
50. Salkin, D., Cadden, A. V., and McIndoe, R. B.: The Blocked Pulmonary Cavity, an Anatomical, Roentgenological and Clinical Study, Am. Rev. Tuberc. **36:** 634-648, 1936; *Ibid.:* Postmortem Bronchography, **36:** 649-662, 1936.
51. Shipman, S. J.: Bronchial Factor in Cavitation, Am. Rev. Tuberc. **37:** 336-345, 1938.
52. Pinner, M.: Healing of Tuberculous Cavities, Am. Rev. Tuberc. **42:** 731-737, 1940.
53. Auerbach, O., and Green, H.: The Pathology of Clinically Healed Tuberculous Cavities, Am. Rev. Tuberc. **42:** 707-730, 1940.
54. Brunn, H., Shipman, S., Goldman, A., and Ackerman, L.: Tuberculous Cavitation and Transpleural Decompression, J. THORACIC SURG. **10:** 485-500, 1941.
55. Goldman, A.: Observations of Tuberculosis Cavities With Special Reference to Transpleural Decompression, Dis. Chest **7:** 205-211, 1941.
56. Shipman, S. J., Rogers, W. L., and Daniels, A. C.: Decompression of Tuberculous Cavities; a Skin Flap Operation for Residual Cavities Following Thoracoplasty, Am. Rev. Tuberc. **44:** 588-595, 1941.
57. Rogers, W. L., Shipman, S. J., and Daniels, A. C.: Flap Drainage of Residual Tuberculous Cavities, J. THORACIC SURG. **12:** 88-97, 1942.
58. Pearson, S. V.: The Pathogenesis of Pulmonary Cavities, Brit. M. J. **1:** 380-381, 1930. Quoted by Vineberg and Kunstler.[6]
59. Brooke, C. O. S.: Excessive Spontaneous Inflation of a Lung Cavity, Lancet **2:** 240-241, 1931.
60. Ameuille, P., Delhomme, and Raust, J.: Le Broncheo de Drainage des Cavernes Tuberculeuses (Etude Anatomique), Ann. d'anat. path. **13:** 849-855, 1936. Quoted by Coryllos and Ornstein.[47]
61. Loesch, J.: Closure of Tuberculous Cavities, Am. Rev. Tuberc. **50:** 500-519, 1941.

62. Thomas, C. P.: Pulmonary Cavities: Their Persistence and Closure, Brit. J. Tuberc. **36:** 4-18, 1942.
63. Meyersburg, H., Gruber, H., and Lupo, C. W.: Bronchoscopic Suction, an Aid in the Closure of Cavities in Pneumothorax Cases, Am. Rev. Tuberc. **45:** 368-372, 1942.
64. Riggins, H., and Gearhart, R. P.: Studies of the Pathogenesis, Dynamics, and Closure of Tension Cavities. Read at the Twenty-fifth annual meeting of The American Association for Thoracic Surgery, Chicago, May 6, 1944. Quoted by Maier.[65]
65. Maier, H. C.: Surgical Treatment of Tension Cavities in Pulmonary Tuberculosis, Am. Rev. Tuberc. **51:** 1-6, 1945.
66. Bernou, A.: The Treatment of "Tension Cavities," Brit. J. Tuberc. **41:** 11-15, 1947. Abst. of Le traitement chirurgical des cavernes "en tension," Rev. de la tuberc. **10:** 343-374, 1946.
67. Bobrowitz, I. D.: Treatment of Tension Cavities With Pneumothorax, Dis. Chest **13:** 133-150, 1947.
68. Brock, B. L.: Discussion of article by Bobrowitz, ref. 74, pp. 150-152.
69. Even, R., and Lecoeur, J.: Les cavernes ballonnisees, inertes et residuelles, Rev. de la Tuberc. **10:** 109-116, 1946; abst. in Am. Rev. Tuberc. **55:** 141, 1947.
70. Le Carboulec, G.: De l'obliteration bronchique provoquee, Rev. de la tuberc., Paris **9:** 201-203, 1945.
71. Hochberg, L. A., Fink, I., and Chardack, W. M.: Cavernostomy, Quart. Bull. Sea View Hosp. **8:** 343-359, 1946.
72. O'Brien, E. J., O'Rourke, P. V., Test, F. C., and Skinner, E. F.: Cavernostomy, J. THORACIC SURG. **16:** 602-622, 1947.

DIAPHRAGMATIC HERNIA

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INTRODUCTION

IN THE past decade outstanding articles have appeared in the literature on diaphragmatic hernia. It is a condition which nowadays is diagnosed clinically with greater frequency than it was some twenty to thirty years ago. Harrington, Hedblom, Truesdale, Ladd and Gross, Marks, Donovan, and Sweet have contributed much in American literature on this subject.

Historically Paré, in 1579, reported the autopsy findings in a case of diaphragmatic hernia caused by trauma. Cooper reported several cases in 1798. Between 1856 and 1920, Rowlands found only two cases in the records of Guy's Hospital in London.

EMBRYOLOGY

The development of the diaphragm and the descent of the stomach, as well as the formation of the pleural and peritoneal cavities, makes the diaphragm susceptible to weak areas through which hernias may occur. They may be present at time of birth or may develop later on in life.

The complex separation of the celomic cavity into pleural, pericardial, and peritoneal cavities begins in the 2.1-mm. embryo. It is completed in the 20-mm. embryo. If a hernia of a portion of the gut occurs before this division is completed, the loops of bowel will not be covered by serous membrane. The hernia would not be a true one. If the hernia occurs after the serous cavities are formed but before the muscular elements of the diaphragm have completely fused, the bowel would be covered by serous membrane. It would then be a true hernia. The communication between the pleural and peritoneal cavities closes in the third month of intrauterine life.

The muscle fibers that grow in between the pleural and peritoneal cavities are derived from the third and fourth cervical myotome, while in the cervical region.

The anterior, lateral, and central portions of the diaphragm develop from the septum transversum and the fused ventral mesentery.

The posterior, lateral, and central portions of the diaphragm are formed by the fusion of the dorsal mesentery and mesoderm with the pleuroperitoneal membrane. This membrane is derived from the pulmonary ridge. Unilateral absence of the diaphragm is probably due to failure of development of the pleuroperitoneal membrane. It is then found as a narrow ridge of tissue along the posterior wall of the thorax.

The mesodermal cells derived from the receeding Wolffian body form the right and left crura of the diaphragm.

Read (by title) at the Twenty-eighth Annual Meeting of The American Association for Thoracic Surgery, Quebec, Canada, May 31, June 1, 2, 1948.

The dorsal mesentery forms the posterior and central portions of the diaphragm. This portion contains the esophageal hiatus.

The pleuroperitoneal membrane grows ventrally. It closes the remaining opening, namely, the pleuroperitoneal hiatus between the pleural cavity and the peritoneal cavity. This closure occurs by fusion between the pleuroperitoneal membrane and the septum transversum. This forms the lateral portion of the diaphragm. The hiatus closes in the 17- or 18-mm. embryo on the right side, and in the 19- or 20-mm. embryo on the left side.

If the mesoderm is not properly deposited at any of these adjacent points of union, or if there is failure of proper fusion, it may result in the congenital continuity of the pleural and peritoneal cavities; or it may result in congenital weak points in the diaphragm. These weak points would occur at points of fusion of the previously named structures.

These weak points would be at:

1. Foramen of Bochdalek—pleuroperitoneal hiatus situated dorso-laterally.
2. The outer crus.
3. The esophageal hiatus.

The stomach descends behind the septum transversum. It should have reached its permanent site before the left half of the diaphragm is closed. If there is a delay in the descent of the stomach, the lumbar portion of the diaphragm will not have developed perfectly and the esophageal hiatus will have developed around the cardia of the stomach instead of around the esophagus. This results then in an unusually large esophageal hiatus. There will be a deficiency in both the muscle ring and in the attachments of the diaphragmatico-esophageal membrane. The abnormal position of the stomach will cause the widening of the esophageal hiatus and a wide space between the margins of the muscle and the wall of the esophagus. It will also cause improper fixation of the elastic membrane between the muscle margin and the stomach and esophagus as well as an abnormally large peritoneal fold extending well down on the cardia of the stomach.

The more defective the development of the esophageal hiatus the greater are the chances that an esophageal hiatus hernia may develop later on in life.

The dome of the diaphragm is not a fusion region. Therefore, any hernias that occur through this portion of the diaphragm cannot be attributed to embryologic development. It must be due to either degeneration of the muscle or some other pathologic condition or injury.

Embryonic and congenital hernias are, however, the result of improper development of parts of the diaphragm. Persistence of the dorsopleural recess may be a factor in the origin of these hernias.

All of these factors explain the fact that diaphragmatic hernias are more common on the left side than on the right side.

ANATOMY

The diaphragm is composed of three parts which insert into a central tendon. These parts are:

1. The sternal portion.

2. The costal portion.
3. The lumbar portion.

The sternal portion arises from the xiphoid process. The costal portion arises anteriorly and laterally from the six lower ribs. The lumbar portion arises posteriorly from the crura and from the two lumbocostal arches.

The crura arise from the first three lumbar vertebrae.

The medial lumbocostal arch of Haller is a fibrous band which extends from the body of the second lumbar vertebra to the tip of the first lumbar transverse process. Under this arch passes the psoas muscle. The lateral lumbocostal arch of Haller extends from the tip of the first lumbar transverse process to the twelfth rib. The quadratus lumborum muscle passes under this arch.

Even in a normal adult there may be a triangular space which separates the muscle fibers of the outer crura of the lumbar portion and the last muscular portion of the costal portions of the diaphragm. This space is filled in by a thin layer of connective tissue, the continuation of the iliac fascia, which is covered above by pleura and below by peritoneum. This layer of connective tissue is less elastic than the surrounding fibers and, therefore, may be more easily torn. This area is protected by the liver and by the fatty tissue surrounding the adrenal and kidney on the right, or else it would more often be the site of hernias. It explains the fact that 95 per cent of all hernias occur on the left side.

Failure of fusion of the lumbar and costal portions in children results in a persistent hiatus in this triangular space or the hiatus pleuroperitonealis. This was described in 1848 by Bochdalek and, therefore, is commonly called the foramen of Bochdalek. The apex is curved toward the central tendon upward and forward. The base is turned downward and somewhat backward and partly rests on the twelfth rib. The borders of the foramen are muscular and are of a somewhat sphincter-like construction.

The inner crus of the diaphragm is separated from the middle crus by slits through which pass:

1. The greater and lesser splanchnic nerves.
2. The vena azygos major on the right, although it may pass through the aortic opening.
3. The vena azygos minor on the left.

These are not sites for hernias.

If there is a failure of fusion of the sternal portion with the costal portion of the diaphragm, an opening exists to either side of the xiphoid process. This opening is filled in with areolar tissue and is covered above with pleura and below with peritoneum. The deep epigastric vessels pass through this space. These openings are called the foramina of Morgagni or Larrey's spaces.

The diaphragm and transversus abdominis muscle are continuous with one another. This can be readily demonstrated by separating them from the costal arch. This converts the two muscles into a continuous fibromuscular sheet with a common aponeurosis. With the rectus sheath and the lumbar fascia this sheet encloses the abdominal cavity (Rives and Barker). The diaphragm is continuous with the inner fibromuscular layer of the abdominal wall.

There are several openings in the diaphragm through which important structures normally pass:

1. Aortic opening.
2. Opening for the vena cava.
3. Esophageal hiatus.

The aortic opening is actually behind the diaphragm at the level of the first lumbar vertebra. The crura form the lateral boundaries as they extend forward from their origin on the anterior aspect of the spine. The opening is bounded by dense fibrous tissue. The azygos vein and the thoracic duct pass through this opening close to the aorta. No hernia has ever been known to occur through this opening.

The vena cava opening is higher and more anterior on the right side. It is at the level of the intervertebral disc between the eighth and ninth vertebrae. It lies within the central tendon and is protected by the liver. It is not the site of hernias.

The esophageal hiatus is more anterior than the aortic opening and is at the level of the tenth thoracic vertebra. It transmits the esophagus, the esophageal vessels, and the vagus nerves. It lies entirely within the muscular portion of the diaphragm. It is surrounded by decussating muscle bundles of the crura before they spread out and insert into the central tendon. The size of the opening varies greatly. The muscle bundles of the hiatus are innervated by the left phrenic nerve. It is normally situated to the left of the midline.

The muscle fibers of the hiatus are only loosely approximated to the lower end of the esophagus and are not directly attached to it. The diaphragmatico-esophageal membrane brings about the fixation of the diaphragm to the lower end of the esophagus and the upper end of the cardia. It is inserted into the lower 2 to 3 cm. of the esophagus and the upper 1.5 to 2 cm. of the stomach in a fanlike manner. It is composed of elastic and fibrous tissue.

The membrane allows a certain amount of mobility between the diaphragm, esophagus, and cardia during swallowing and respiration. It acts as an antagonist to the longitudinal muscle fibers of the esophagus which tend to pull the cardia through the hiatus.

The abdominal portion of the esophagus is loosely covered to the left and anteriorly by peritoneum. The uppermost portion of the stomach is loosely covered by peritoneum for 2 cm. anteriorly and 3 cm. posteriorly. Here the peritoneum fuses with the serous covering of the stomach. The union of these two folds forms the gastrophrenic ligament which fixes the cardia of the stomach to the diaphragm. The ligament continues to the left to form the phrenico-colic ligament. Under this loose peritoneum is a fat pad. This loose peritoneal attachment allows free motion of the esophagus without traction on the peritoneum (Harrington).

CLASSIFICATION

There is no universally accepted classification of diaphragmatic hernia. The two best classifications are the one by Harrington and the one by Marks.

Harrington classifies the hernias into:

1. Nontraumatic.
2. Traumatic.

The nontraumatic hernias are again divided into:

1. Congenital—at birth.
   a. through foramen of Bochdalek
   b. esophageal hiatus
   c. through foramen of Morgagni
   d. through absence of the diaphragm
2. Acquired after birth.
   a. through esophageal hiatus—with sac
   b. through region of the fusion anlages of the diaphragm
   c. through the sites of congenital hernia

The traumatic hernias are divided into:

1. Indirect—by crushing injury.
   a. at any point of the diaphragm
   b. through the esophageal hiatus—usually with sac
   c. through the leaf of the diaphragm—usually no sac
2. Direct.
   a. by missile or knife
   b. rupture due to
      1. necrosis—abscess below diaphragm
      2. inflammatory necrosis in empyema with pressure necrosis of drainage tube usually in the posterior diaphragm with no sac.

Marks's classification is also a useful one. It is based on his findings in x-ray studies of 29 cases over a period of two and one-half years. He bases it on whether the cases are amenable to surgical repair or not.

His classification is:

1. Thoracic stomach with very short esophagus and entire stomach above the diaphragm.
2. Diaphragmatic hernia with short esophagus, part of the stomach above the diaphragm.
3. Hiatus hernia—esophagus of normal length, hernia through hiatus composed of stomach, possibly the colon.
4. Congenital hernia—
   a. through the foramen of Bochdalek
   b. through the foramen of Morgagni

   containing large and small bowel, frequently with other organs.
5. Congenital absence of diaphragm.
6. Eventration of diaphragm.
7. Traumatic hernia.

The type of hernias that require surgical intervention are:

1. Esophageal hiatus hernia.
2. Traumatic—direct or indirect, inflammatory, or necrosis of the diaphragm.

3. Absence of the diaphragm.
4. Hernia through the hiatus pleuroperitonealis—foramen of Bochdalek.
5. Hernia through the foramen of Morgagni.

Signs of hernias that require surgical intervention are:

1. Cases in which the stomach alone is involved. When these hernias get bigger, the omentum and colon also enter the hernia as well as the spleen.
2. Cases in which multiple organs are involved as the traumatic or congenital hernias. The symptoms may become more varied and severe, and there may be development of obstruction or severe hemorrhage.

THORACIC STOMACH

Bailey described this condition in 1919. It is not acquired but is a congenital abnormality. It is not amenable to surgery. All or nearly all of the stomach is above the diaphragm, fixed by reason of an extremely short esophagus. It is really not a hernia. The stomach never was in a normal position. To be a hernia the organ must have been in a normal position and then shifted into an abnormal position. The esophagus enters the top of the stomach and may be only three inches long. The pylorus lies at the hiatus, above or just below the diaphragm. The esophagus may end at the level of the fourth thoracic vertebra.

DIAPHRAGMATIC HERNIA WITH SHORT ESOPHAGUS

1. The stomach is surrounded by a serous sac.
2. The esophagus ends at the level of the seventh or eighth thoracic vertebra.
3. The stomach is in the posterior mediastinum.
4. It is important to differentiate as surgical repair is possible but is very difficult.
5. The difference between normal and abnormal length of the esophagus may be only a matter of centimeters.
6. The upper end of the stomach looks like a dilated esophagus, and there is no evidence of a rounded fundus.
7. The esophagus enters the stomach at the uppermost portion.
8. In a true hiatus hernia the esophagus is not shortened but is tortuous. The entrance into the stomach is to one side or other, not in the midline at the apex of the stomach.
9. The hiatus hernia is sometimes only seen on x-ray examination with the patient recumbent, supine, or in Trendelenburg posture. In a shortened esophagus the stomach is at all times within the chest even with the patient erect.
10. The stomach must remain at all times partially within the chest.
11. A hiatus hernia is only visualized in the erect posture if large or if the structures are adherent.
12. Frequently, a stricture is present at the junction of the esophagus with the stomach, and there may be some difficulty in swallowing.
13. A shortened esophagus may be treated surgically in some instances.

14. Harrington has reported cases in which the left diaphragm was paralyzed and the hiatus could then be closed around the esophagus above the stomach. It depends a great deal upon the symptom complex whether surgical repair should be contemplated and undertaken.

HIATUS HERNIA

The esophageal hiatus hernias may be divided into:

1. Small hernias with the pouch of herniated stomach alongside the esophagus, the so-called paraesophageal hernias. The esophagus is below the diaphragm. These cases formed one-third of all the patients operated on by Harrington.

2. Large hernias in which the esophagus lies above the diaphragm but is not short. The stomach extends into the mediastinum and may fill the mediastinum or it extends either into the left chest or possibly the right chest. These cases formed two-thirds of the cases operated on by Harrington.

3. Very small pulsion types of hernias due to the incompetency of the hiatus. This occurs in older persons due to atrophy of the diaphragmatico-esophageal membrane.

With younger persons, if the hiatus is abnormally large, a hernia may develop with increased intra-abdominal pressure, respiratory difficulties, or by trauma. In older persons with abnormally large hiatus and with atrophy of the diaphragmatico-esophageal membrane hernias may develop. These belong to Group 3 above.

In the smaller types of hernia the elastic membrane forms one of the coverings of the hernial sac. In larger hernias the membrane is pushed forward and the hernial sac enters the posterior mediastinum without any barrier. The loose peritoneal fold fuses with the stomach serosa farther down than normal in true hernias. The peritoneal fold and the gastrophrenic ligament join the cardia of the stomach in an abnormal manner when the hiatus muscular ring is abnormally enlarged. These hiatus hernias, therefore, are probably the result of delayed descent of the esophagus in relation to the development of the lumbar portion of the diaphragm.

The smaller hiatus hernias may turn into the larger hernias similar to other types of sliding hernias. Occasionally, the lower end of the esophagus may dilate.

Hiatus diaphragmatic hernias are the most common type. They must constantly be considered in the differential diagnosis of upper abdominal pathology. The large hiatus hernias must be differentiated from the thoracic stomach with a short esophagus. If a good part of the stomach passes up through the hiatus, the gastrocolic omentum may drag a portion of the colon up with the stomach into the hernial sac. The stomach may frequently be inverted. It usually lies to the left of the midline. The intrathoracic portion usually rotates with the axis passing through the cardia so that the greater curvature lies cephalad.

Symptoms.—These may be vague. The size of the hernia does not have much relation to the symptoms. Small hernias may give severe symptoms.

The symptoms may depend on the time of examination and the stage and size of the hernia. Ohler and Pitko have described a hiatus hernia syndrome:

1. Feeling of pain, fullness, or distress in the epigastrium and under the tip of the xiphoid coming on at the beginning of a meal. It may be so severe that the patient has to stop eating.
2. When the patient gets up and walks about the symptoms suddenly disappear and the patient can continue eating the meal.

This syndrome practically permits a diagnosis of a hiatus hernia. Cardiospasm and esophageal diverticulum can give similar symptoms on beginning a meal, but when the patient walks about the symptoms do not disappear as quickly.

The coexistence of other intra-abdominal conditions must constantly be kept in mind. There is a high incidence of coexistence of duodenal diverticulum and colon diverticulosis. Weintraub and Tuggle in 310 cases of duodenal diverticulum found a coexistence of 9 per cent of hiatus hernia and 23 per cent of colon diverticulosis.

Pain, distress, eructation, vomiting, dyspnea, heart palpitation, and weakness are frequent symptoms. These symptoms may be very severe with pain radiating down the left arm. Some of these symptoms may be caused by pressure on the vagus nerve in the esophageal hiatus. These symptoms may be relieved as mentioned previously in the syndrome findings by assuming the upright position or by lying down, by soda bicarbonate, by induced vomiting, by belching, or by defecation. Hemorrhage with secondary anemia are complications which are quite common. Erosions, ulcers, and varicosities of blood vessels occur in the herniated portion of the stomach.

Differential Diagnosis.—The main diseases from which hiatus hernia must be differentiated are:

1. Gastric or duodenal ulcer.
2. Cholecystitis with or without cholelithiasis.
3. Hyperacidity or secondary anemia.
4. Cardiac disease, especially angina pectoris.
5. Carcinoma of the cardia, stricture and diverticulum of the esophagus.
6. Appendicitis.
7. Intestinal obstruction.

One must constantly be conscious of the possibility of the presence of a hiatus hernia in addition to any one of the preceding conditions.

Physical Signs.—In small or medium hiatus hernias physical signs are absent. Only in the large hernias are signs present and then may be present or absent at various times of examination. There may be asymmetry of the chest with widening of the intercostal spaces. Percussion may elicit shifting tympany and dullness. Auscultation may show gurgling, tinkling, or a splashing sound in the chest region to the left or right or both sides.

Diagnosis.—The roentgenologist will be invaluable with most careful study and examination, without contrast medium, with barium, and by fluoroscopy, and may help in determining the method and course of treatment. Seidlitz powders will distend the stomach with gas and may be of material aid.

Treatment.—Treatment depends a great deal on the size of the hernia. For small and medium cases conservative treatment is indicated. For large hernias, if more than one-third of the stomach has herniated, and especially those containing colon, surgery is indicated. The danger of intestinal obstruction is ever present. Fixation of the stomach to the diaphragm or to the hernial sac causes much difficulty in treatment. Fortunately, such complications are rare. Early surgical repair is, therefore, indicated.

Many papers on the surgical treatment of hiatus hernias have been published. Harrington has been one of the main contributors. A brief summary of these papers is as follows:

A. Palliative surgical treatment.

Temporary or permanent interruption of the left phrenic nerve is of help in many of the small or medium hiatus hernias. It may prevent spasm in small hernias.

B. Radical surgical repair of hiatus hernias.

1. Temporary or permanent interruption of the phrenic nerve is indicated in many cases. Temporary crushing of the nerve will result in restoration of function in from three to six months. If the abdominal approach is planned it may be a wise preliminary step.
2. Intratracheal anesthesia with use of positive pressure is essential.
3. *Approach.*
   a. Abdominal approach. Harrington prefers the abdominal approach except in large hernias on the right side. He feels that the abdominal approach either through a subcostal or left rectus incision is preferable to the thoracic approach as the hernial contents are usually contained within a hernial sac in the posterior mediastinum. The hernial contents are not actually within the pleural cavity. The esophageal hiatus is to the left. He feels that there may be less complications than in a thoracic approach.
   b. Thoracic approach. Personally we agree with Sweet that the thoracic approach in the majority of cases makes the surgical technique somewhat easier. The hernial sac, the esophageal hiatus, the cardia of the stomach lie directly before one. The phrenic nerve can be crushed just above the entrance into the diaphragm at the beginning of the operation putting the diaphragm at rest. The freeing of the sac from the hernial aperture and the cardia of the stomach is simplified. The reduction of the hernial contents is easy. In very large hernias in which the reduction is more difficult Sweet has suggested making a separate counter incision in the diaphragm, reducing the hernial contents and temporarily drawing them through this counter incision over the lower edge of the chest wall wound until the hernial aperture has been closed. The objection of painful postoperative thoracic wounds has not been observed in sufficient numbers to be offered as a contraindication to the thoracic approach.
4. In the abdominal approach the left lobe of the liver must be freed by cutting the suspensory ligament and either turning the lobe to the right, or bringing it forward into the wound, thus giving better access to the hiatus. The spleen must either be separated or, if necessary, removed.
5. The sac of the hernia must be completely separated from the stomach. Its edges must be separated from the hiatus opening. The sac must be either totally removed or allowed to retract into the chest or mediastinum or plicated and dropped into the abdominal cavity as suggested by Sweet.
6. Closure of the defect. It is well to pass a large stomach tube into the stomach at the time of closing the defect so that it will not be closed too tightly and cause difficulty in swallowing. One should be able to insert the tip of the index finger between the esophagus and the closed defect. The repair should preferably be either to the left

or behind the esophagus. Overlapping repair of the muscle edges should be accomplished with silk or cotton sutures. Interweaving of living fascia lata obtained during operation by an assistant is of great value in difficult cases as advised by Harrington. The esophagus may be incorporated in the suture. Great care must be exercised not to perforate the stomach if erosion or ulceration exists.

7. Exploration of the abdomen for other conditions is advisable if the abdominal approach has been employed. Immediate attention to abnormal findings, however, may not be advisable. Operations for secondary pathological findings should only be performed if absolutely essential.

8. Recurrence of esophageal hiatus hernias is not uncommon. Sweet has shown that recurrence seems to be much less frequent at the Massachusetts General Hospital following thoracic approach and repair than following the abdominal approach. If a recurrence should occur following repair by the abdominal approach the secondary operation should always be by the thoracic approach. If, therefore, recurrences are less frequent by the primary thoracic approach this method seems to be the method of choice.

CASE REPORT

CASE 1.—A young girl, E. L., 23 years of age was admitted to the Lenox Hill Hospital in November, 1947, with a complaint of nausea, vomiting, epigastric pain of three years' duration. These symptoms began three years previously and occurred in the evening at the end of a day's work. Vomiting gave relief. The attacks became more frequent until they occurred several days in a row. The attacks never occurred if the patient stayed in bed or was lying flat. Roentgenograms were taken three years previously and a diagnosis of hiatus hernia made at that time. The patient had lost fifteen pounds in the year previous to admission.

Gastric analysis showed free hydrochloric acid of 38 and total acidity of 46. The string test, after a white diet, showed a positive blood stain. There was a secondary anemia of 3,300,000 red blood cells, 9.6 Gm. hemoglobin or 64 per cent, 6,900 white blood cells with 66 per cent filaments and 9 per cent nonfilament polymorphonuclear leucocytes, 21 per cent lymphocytes, 2 per cent monocytes, and 2 per cent eosinophiles. The sedimentation rate of the blood was normal. The weight of the patient was 115 pounds.

X-ray Examination.—The liver was normal in size and its position was right-sided. The hepatic flexure of the colon was outlined by gas and was slightly low in position. X-ray examination of the chest showed the heart and aorta to be normal in size and contour. Irregular linear shadows were seen over the lower third of the right lung field, indicating the presence of stomach or intestine in the right thorax. The right leaf of the diaphragm could not be made out. The lungs were clear and well aerated throughout (Fig. 1).

Alimentary Tract.—Examination of the alimentary tract by means of a barium meal demonstrated that the entire stomach projected through a hiatus type of diaphragmatic hernia, the opening of which was probably not more than two inches in diameter. The entire stomach occupied the lower third of the right thorax posteriorly. The duodenal loop was in its normal position. At six hours only the traces of barium remained in the stomach. The remainder of the meal progressed at an average rate. The course of the small and large intestine was normal, the cecum being situated to the right, but the distal portion of the transverse colon was contained within the diaphragmatic hernia (Figs. 2 through 5).

Preoperative Preparation.—The patient was prepared with penicillin preoperatively, 30,000 units every three hours. Low residue diet was given. A Levine tube was passed and the stomach lavaged with soda bicarbonate and Wangensteen suction.

Operation.—On Nov. 25, 1947, the operation was performed using intratracheal anesthesia, gas, oxygen, ether, and cyclopropane.

In this instance on account of the extensive right-sided thoracic hernial contents it was decided to employ an abdominal approach. (Technical difficulties during the operation later proved this to be an unwise decision.) Therefore a left oblique per rectus incision was made

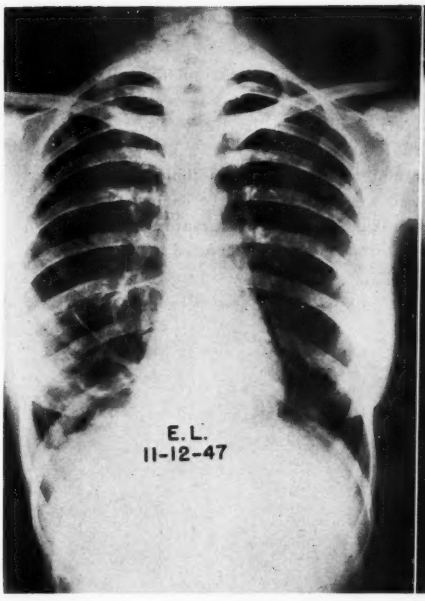

Fig. 1.

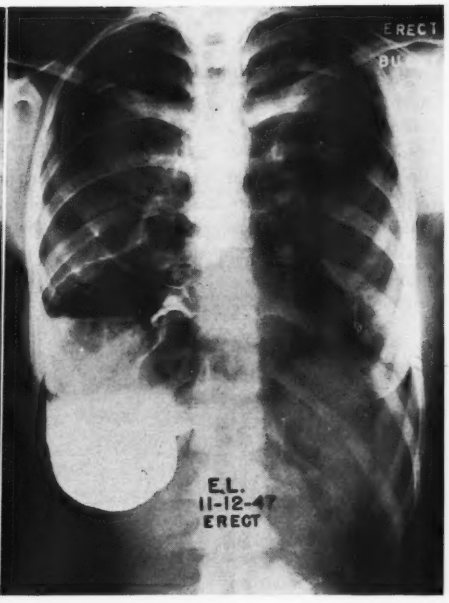

Fig. 2.

Fig. 1 (E. L.).—X-ray view of chest before barium meal. Esophageal hiatus hernia.
Fig. 2 (E. L.).—Barium meal showing stomach in right thoracic cavity. Erect posture.

Fig. 3.

Fig. 4.

Fig. 3 (E. L.).—Oblique view.
Fig. 4 (E. L.).—Oblique supine view showing stomach high up in right thoracic cavity.

from the ensiform cartilage downward and outward to the level of the umbilicus. The abdominal cavity was entered. The stomach was not seen. The colon was seen passing into the right chest through the hiatus opening. The left lobe of the liver was freed by dividing the suspensory ligament and, thereby, exposing the hiatus opening. With a stick sponge in the hiatus opening air was sucked into the hernial sac making reduction of the hernial contents easier. The colon was withdrawn first and was followed by the entire stomach and omentum. The stomach had rotated 180 degrees so that the greater curvature was cephalad and the lesser curvature caudad. Step by step the hernial sac was separated from the cardia anteriorly, laterally, and posteriorly, exposing the esophagus. This procedure was more difficult especially on the posterior aspect of the stomach through the abdominal approach.

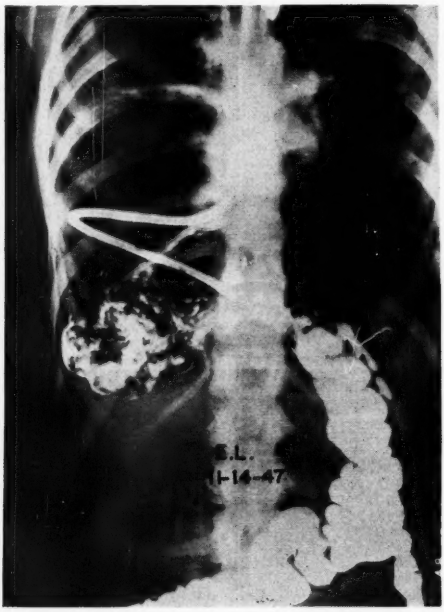

Fig. 5 (E. L.).—Forty-eight hour view. Barium in transverse colon within right thoracic cavity. Note Levine tube in stomach.

Transthoracic access would have been much simpler. Hereupon, the parietal portion of the hernial sac was separated from the margin of the hiatus opening and the two widely separated crura of the diaphragm overlying the aorta. The peritoneum of the hernial sac was then allowed to recede upward into the right chest cavity. A large stomach tube was introduced into the stomach, and the hernial aperture then repaired. The hiatus opening was closed around the esophagus employing silk mattress sutures—three ventral to the esophagus, and two dorsal to the esophagus. Here the tissues were poor in quality. After tying of the sutures, it was felt that the esophageal opening was too tight. Therefore, one ventral silk suture was removed and replaced with a cotton suture. Cotton sutures were then employed to reinforce the silk mattress sutures. The left lobe of the liver and the spleen fell into place and came up to the repaired hiatus opening. The stomach and colon were then placed in the proper position. The stomach was found to be unusually large and filled a large portion of the left part of the abdominal cavity. The abdominal wall was closed in layers employing interrupted silk sutures.

The patient received 500 c.c. of blood and 1,500 c.c. of saline with glucose during the operation. She left the operating room in good condition.

Postoperative Course.—The patient made an uneventful surgical convalescence. However, a gastrointestinal x-ray examination on the thirteenth postoperative day showed a very large atonic stomach within the abdomen. There was marked 24- and 48-hour gastric retention (Figs. 6 and 7). The barium immediately passed through the pylorus at the time of examination and filled the duodenum. There was practically no evidence of peristaltic

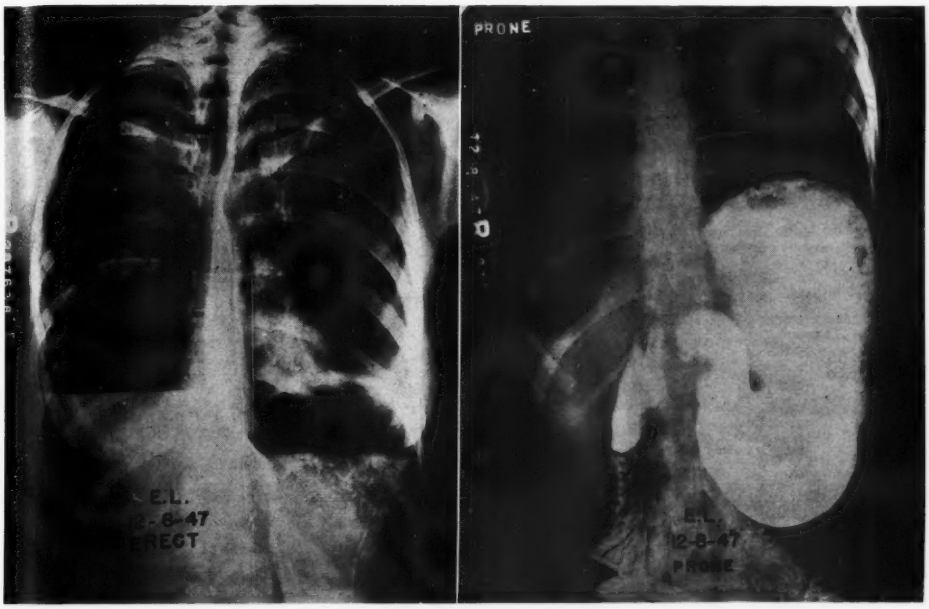

Fig. 6. Fig. 7.

Fig. 6 (E. L.).—X-ray view of chest postoperative showing fluid level in empty hernial sac within right thoracic cavity. Note barium passing normally into stomach within abdominal cavity.

Fig. 7 (E. L.).—Showing large atonic stomach filling left side of abdominal cavity.

activity in the stomach. At 6 hours practically 75 per cent of the barium was retained in the stomach. The remainder of the meal was progressing normally. At 24 hours and 48 hours there was at least 50 per cent retention in the stomach. A gastric analysis showed a total absence of free hydrochloric acid with a total acidity of only thirty. Fractional gastric analysis with histamine performed on December 16 showed a total absence of free hydrochloric acid over the 90-minute period. The total acid went up to 40 at the end of one hour and dropped to 20 at 90 minutes. A Hollander insulin test showed an absence of free hydrochloric acid with a total acidity of 16 with the blood sugar dropping to 22 mg. per cent at 30 minutes and rising again to 83 mg. per cent at 2 hours. The possibility of injury to both vagus nerves at the time of separation of the peritoneum from the cardia of the stomach was considered.

The patient was discharged from the hospital on December 20. Early in January she was put on Urecholine as a trial to see whether the atony of the stomach would improve. At the end of January x-ray studies were repeated (Fig. 8), and there was still evidence of gastric atony but to a lesser degree. There was still 24-, 48-, and 72-hour retention. The

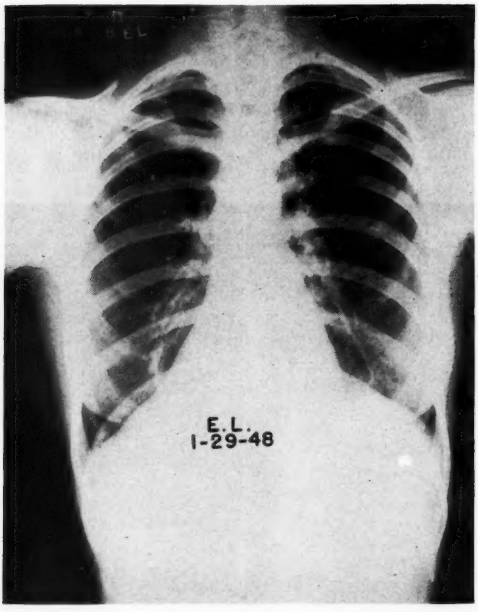

Fig. 8 (E. L.).—X-ray view of chest two months after operation showing complete absorption of fluid from hernial sac in right thoracic cavity.

Fig. 9. Fig. 10.

Fig. 9 (E. L.).—Showing large atonic stomach 4 months after operation.
Fig. 10 (E. L.).—Showing 48 hour residue in stomach 4 months after operation.

stomach was well within the abdominal cavity, but a very small pouch of the stomach protruded upward through the esophageal hiatus. The peritoneal sac in the right chest did not show the fluid level and air within the sac which had been seen postoperatively. The patient was doing well, but gastric lavage was necessary to keep her comfortable in an attempt to improve the condition of the gastric musculature.

The patient was readmitted to the hospital for study in April and June, 1948 (Figs. 9 and 10). Each time there was evidence of increasing free hydrochloric acid on gastric analysis with histamine, and the total acidity had also increased. The Hollander insulin test showed the usual drop in blood sugar but not a concomitant rise in free hydrochloric acid. X-ray examination showed the size of the stomach to be smaller, but there was still marked atony with very little evidence of peristalsis. It is interesting to note in this case that the patient to a degree had adjusted herself to the presence of the stomach within the right chest cavity. After replacement of the stomach into its normal position within the abdominal cavity, it was found that the stomach did not function as normally as one had hoped it would. In July, 1948, the patient was feeling very well, gaining weight, and worked a full day. Only occasionally did she lavage her stomach.

In retrospect a thoracic approach would have been preferable in the repair of this hernia. Access to the enlarged esophageal hiatus would have been easier. Separation of the peritoneal hernial sac from the cardia of the stomach especially posteriorly and from the margins of the hernial aperture would have been much easier. Crushing of the phrenic nerve intrathoracically would have been advantageous. The apparent injury to vagus nerve fibers with marked postoperative gastric atony might have been avoided. The convalescence would have been markedly simplified thereby.

CONGENITAL HERNIA

Historical.—Ladd and Gross, Harrington, Truesdale, Donovan, Dorsey, and Marks have written extensively about congenital hernias. Orr and Neff, in 1936, found only 17 patients operated in the first year of life. Of these only 9 survived. There is little to recommend delay.

Types.—1. The commonest hernia occurs through the pleuroperitoneal hiatus or the foramen of Bochdalek. 2. They may also occur through the esophageal hiatus. That the latter is the commonest type in adult life is due to the fact that babies with congenital hernias through the foramen of Bochdalek have had such a high mortality. 3. The third type are the congenital hernias through the foramen of Morgagni or the substernal hernias. These hernias are more common on the right side.

The hernias through the foramen of Bochdalek have no hernial sac while those through the foramen of Morgagni have a hernial sac.

Symptoms.—The symptoms are severe. Death is frequently caused by asphyxia or intestinal obstruction. Respiratory, circulatory, or gastrointestinal, or a combination of these three main symptoms may be present. If there is dyspnea, cyanosis, or vomiting during crying or nursing following delivery, immediately suspect a congenital diaphragmatic hernia.

Physical Signs.—

1. Rapid respiration and pulse rate.
2. Heart displaced from the affected side.
3. Tympany or dullness on percussion of the affected side.
4. Absent or distant breath sounds on auscultation, also the presence of intestinal gurgles.

5. Scaphoid abdomen as most of the abdominal contents are in the chest, especially the small and large intestines, usually with nonrotation of the colon. The spleen, omentum, and possibly portions of the liver may be in the chest.

Diagnosis.—The suspicion of the presence of diaphragmatic hernia is the main diagnostic feature. X-ray evidence is of value, preferably without contrast medium.

Treatment.—It is the consensus of those authors who have had greatest experience such as Ladd and Gross, Donovan, and Harrington that the earlier the repair is performed the better the chance of survival of the infant. The abdomen does not develop properly without its proper contents, if the hernial contents are permitted to remain in the chest. While some few infants may accommodate themselves to the presence of the abdominal contents in the chest, their later return to the nondeveloped abdomen becomes almost an impossibility. Therefore, the safest and wisest procedure is surgery as quickly as the diagnosis is made. Newborn infants stand major surgical procedures better than they do a week or more after birth. The youngest infant operated by Ladd and Gross was only forty hours old. The first forty-eight hours are the best, the intestines are not as distended and can be returned more easily into the abdominal cavity.

Preoperative Preparation.—

1. Hydration.
2. Enemas to deflate the bowel.
3. Gastric suction.
4. Oxygen tent with 95 per cent oxygen.

Anesthesia.—Cyclopropane with positive pressure by intratracheal tube.

Operation.—

1. Preliminary phrenic crush in the neck.
2. Almost invariably the abdominal approach for the repair of the hernia.
3. Incision—vertical or subcostal.
4. Allow air to enter the chest through a catheter and then contents can be more easily withdrawn.
5. Denude the edge of the hernia ring of the serous membrane.
6. Suture the opening in the diaphragm with two rows of silk or cotton mattress sutures.
7. Insert a catheter into the chest before closing the last suture so that the air can be sucked out of the chest in order to expand the collapsed lung before tying the last suture.
8. The physiology of respiration is affected by the repair of the diaphragmatic hernia. The sound lung is compressed by increased intra-abdominal pressure on returning the intestinal contents to the abdomen before the bad lung has had a chance to expand fully.

Therefore, if any difficulty is encountered on returning the intestinal contents into the abdominal cavity, Ladd and Gross have proposed a two-stage operation. They believe that their mortality has been greatly reduced by this

procedure. They cut the rectus muscle transversely after under-cutting the skin in order to be able to slide the abdominal wall across the intestinal contents by only suturing the subcutaneous fascia with interrupted mattress sutures and then closing the skin. After the abdominal wall has stretched sufficiently, the incisional hernia is repaired. This is done about five or six days after the first operation. Dorsey stretched the abdominal wall manually and used drop ether anesthesia for deeper relaxation. Ladd and Gross feel that the two-stage operation is preferable to a difficult one-stage closure for the following reasons:

1. It is less shocking.
2. It avoids strain on the suture line of the diaphragm.
3. It avoids postoperative embarrassment of respiratory and circulatory systems.

Harrington advises an x-ray examination in the operating room at the termination of the operation to make sure that the mediastinum is in the center after the air has been withdrawn through the catheter during the operation. If the mediastinum is not in the center, more air must be withdrawn from the chest before returning the infant to the ward.

Postoperative Care.—

1. Oxygen tent relieves respiration and abdominal distention.
2. Fluid requirements parenterally with small transfusions in order not to embarrass the right heart.
3. Fluids by mouth.
4. Watch for pleural effusion.

CONGENITAL ABSENCE OF THE DIAPHRAGM

This is a rare condition. Sagal, in 1933, collected only twelve cases. Dr. William J. Mayo operated on Beckman's case and proved its presence by operation. The positive diagnosis can be made only by operation and determination of the condition by a competent surgeon.

The defect extends from the eighth rib in the posterolateral portion of the diaphragm and extends medially to the esophageal hiatus. There is usually no sac. The opening is so large that the stomach, spleen, large and small intestines have passed through the aperture into the chest.

Treatment of these conditions is more difficult than hernias through the foramen of Bochdalek. It requires great surgical technical skill to bring the diaphragm to the chest wall or the chest wall to the diaphragm. The use of the perirenal fascia or the latissimus dorsi muscles (Rives and Baker) in the repair of the posterior defects is of value. Thoracoplasty with resection of one or more of the lower ribs is occasionally also required. Permanent interruption of the phrenic nerve is necessary.

CONGENITAL SUBCOSTAL-STERNAL HERNIA THROUGH FORAMEN OF MORGAGNI

This is a direct hernia through a congenital defect by failure of fusion of the sternal and costal portion of the anterior diaphragm.

There is always a sac as in an esophageal hiatus hernia. It is the rarest type of hernia while the esophageal hiatus hernia is the most frequent.

The contents are usually colon, omentum, or intestine, rarely the stomach. These pass through a congenital weakened defect later in life due to increased intra-abdominal pressure.

The symptoms are pain, cough, dyspnea, and occasionally intestinal obstruction. The physical findings and x-ray films may make one suspicious of a primary pulmonary lesion, especially if only omentum is in the hernial sac. The lesion being anterior, lateral x-ray films will help one to determine the possibility of this type of hernia if the pathology is close to the diaphragm and anterior.

Treatment of this type of lesion is by surgery. Abdominal approach is by far the best. An oblique rectus incision is advantageous. The round ligament of the liver can be incorporated in the repair if the hernia is on the right side. If the opening is large, the phrenic nerve should be interrupted. The transversus abdominis muscle, which is normally continuous with the diaphragm muscle, may be employed in the repair of the defect.

CONGENITAL SHORT ESOPHAGUS WITH PARTIAL THORACIC STOMACH

The repair of this condition has been reported by Harrington. Permanent interruption of the phrenic nerve is necessary in order to be able to close the diaphragm above the cardia and around the esophagus. By freeing the esophagus thoroughly, the esophagus can be brought down some centimeters.

EVENTRATION OF THE DIAPHRAGM

This is a rare condition more common in men than in women. It is more common on the left side than on the right side, but may be bilateral especially in cases of megacolon. The condition may be acquired or congenital and is caused by aplasia or atrophy of the muscle fibers of the diaphragm.

In 1921, Korns published a comprehensive analysis of proved cases.

The diagnosis is made by the roentgenologist. The differential diagnosis is between a hernia with a sac or eventration of the diaphragm. An arched line above the organs may be either.

The diaphragm may be demonstrated by a pneumoperitoneum. The loops of bowel are drawn together more in a hernia than in an eventration. Symptoms are much less severe in eventration than in a hernia. There is frequently a feeling of pressure in the left chest caused by gas in the stomach above the level of the cardiac orifice. The heart is usually found on the right-hand side. Symptoms like this frequently occur after phrenic crushing in tuberculosis.

TRAUMATIC DIAPHRAGMATIC HERNIA

These are always false hernias without sacs as the diaphragm is torn by penetrating injuries, crushing injuries, or falling injuries. In World War II there were many injuries of the diaphragm caused by shell fragments, bullets, or other missiles. They may occur anywhere in the diaphragm, but the very great majority are on the left side. The diaphragm is frequently torn from the dome all the way to the periphery. It is comparable to an evisceration of the abdominal contents into the pleural cavity. The stomach and large and

small intestines may have herniated through the opening, and signs of intestinal obstruction may be among the first symptoms. The symptoms are severe and progress rapidly. There is interference with the function of the lungs and heart. Adhesions develop along the margins of the hernial orifice.

Treatment is always by surgery. The best approach in this case is transthoracic as it gives better access and makes separation of adhesions easy. The diaphragm can be paralyzed by intrathoracic crush of the phrenic nerve. The margins of the hernial aperture must be debrided and then repaired by careful overlapping suture. Cotton or silk may be used, but living fascia lata is of value if the operation is one of election in a hernia present some months after injury or following inflammatory or pressure necrosis. The diaphragm may be sutured to the intercostal muscle, preferably to two intercostal spaces, if the tear goes all the way to the periphery. Lower thoracoplasty of the eighth, ninth, and tenth ribs should be kept in mind if this is indicated in the proper closure and repair of the diaphragm.

Negative pressure must be re-established by withdrawing air through a catheter, and air-tight underwater drainage is wise especially if contamination of the pleura has taken place. Antibiotics and chemotherapy also are of great value. Aspiration of air from a pneumothorax is important until negative pressure exists.

The replacement of the abdominal organs from the pleural cavity must be orderly. If obstruction exists, an appendicostomy or colostomy may wisely be added. Wangensteen suction of the stomach is required.

Intratracheal anesthesia with positive pressure is essential.

Postoperative oxygen is necessary.

CASE REPORTS

Case reports of three traumatic diaphragmatic hernias are appended:

CASE 1.—A sergeant was admitted to an evacuation hospital two and one-quarter hours after having been hit by a shell fragment. The wound of entrance was in the left posterior axillary line over the tenth rib. It measured about 4 cms. in length. Omentum presented itself in the wound of entrance, having prolapsed through the diaphragm and pleural cavity into the wound of entrance. X-ray examination showed a shell fragment in the region of the left lobe of the liver.

After proper preoperative therapy, the soldier was operated upon under intratracheal anesthesia. The superficial wound was debrided and the ninth rib resected. The pleural cavity was filled with blood, and the lower lobe of the left lung was lacerated. It was repaired by suture after debridement.

The diaphragm was badly torn, and a torn, bleeding spleen had herniated through the hole in the diaphragm. The eighth rib was then resected for better access. The diaphragm was debrided and then incised medially and laterally for better access to the abdominal cavity. The spleen was removed. A 4-cm. rent in the posterior wall of the stomach was closed near the greater curvature. The wound of exit on the anterior wall of the stomach near the lesser curvature was then closed. The shell fragment could be palpated just under the surface of the dome of the left lobe of the liver near the midline. The foreign body was removed from the liver, and the cavity in the liver was drained bringing the drain out through a stab incision made in the anterior abdominal wall. No further damage could be found within the abdomen.

The diaphragm was then repaired with interrupted overlapping sutures of the leaves with silk sutures. The left phrenic nerve had been crushed. The pleural cavity was irrigated with saline, and 40,000 units of penicillin were instilled. Air-tight underwater drainage was instituted. The chest wall was closed and air withdrawn; 3,000 c.c. of blood in Alsever's solution were given during the operation. A Levine tube had been introduced into the stomach on admission and was left in place with Wangensteen suction.

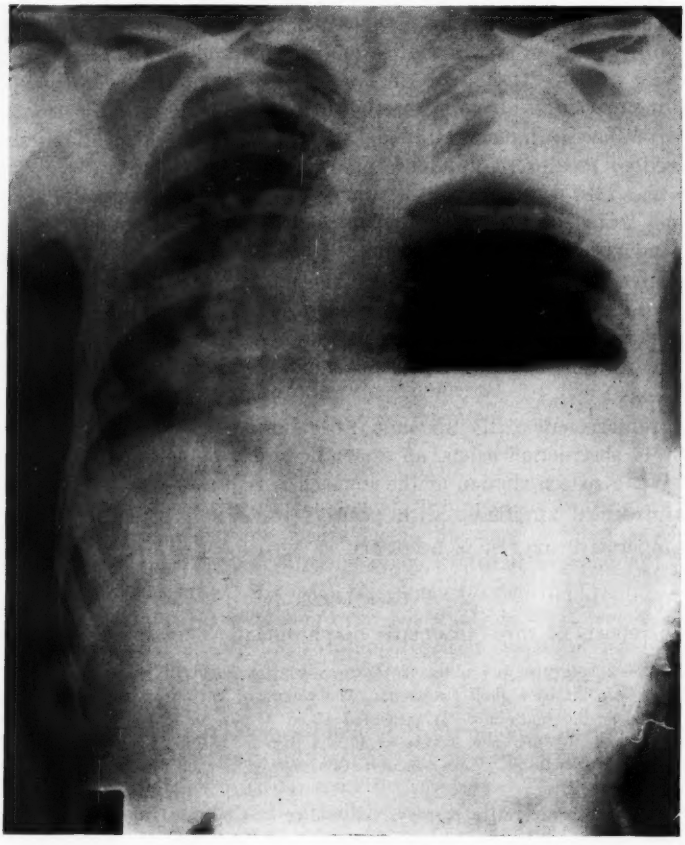

Fig. 11 (J. C., Case 2).—Traumatic diaphragmatic hernia. Note tremendously dilated stomach filled with fluid in left pleural cavity. Heart and mediastinum shifted to right. Patient admitted to hospital in shock.

The patient did well and the pleural drain was removed in 48 hours and the skin wound closed with sutures. Penicillin and sulfadiazine were continued after the operation.

The anterior abdominal drain was changed on the tenth day postoperatively. Fluid was aspirated once from the left pleural cavity. The patient was evacuated on the twenty-second postoperative day. He finally reached England where he was seen by Col. Harken. All wounds were healed six weeks postoperatively.

Three months after his injury he was returned to general duty. While the patient was waiting in the replacement depot the chest wound reopened. He was then operated on for empyema and returned to the United States. At Camp Shanks an abscess of the abdominal

drainage wound was opened. While in the Brooke General Hospital, both wounds healed. While the patient was home on furlough in Rochester, Minn., the chest wound reopened. Later, at operation at Brooke General Hospital, two silk sutures used at the original operation for repair of the diaphragm were found in the abscess cavity. Hereupon, the wound healed and has remained healed. The traumatic diaphragmatic hernia has also remained healed.

Fig. 12 (J. C.).—Lateral view of chest showing similar findings.

CASE 2.—A soldier, private, 19 years of age, was admitted with a diagnosis of perforating gun-shot wound of the left chest wall and diaphragmatic hernia. The wound of entrance on the anterior left lower chest and the wound of exit in the posterior axillary line lower down had been debrided at a more forward installation and had been covered with petroleum jelly gauze. The soldier was admitted in shock, which had developed in transport. Pulse was 150, heart shifted to the right, there were absent breath sounds in the left chest. X-ray examination showed a huge fluid level in the left chest (Figs. 11 and 12). The patient had been evacuated by ambulance without a Levine tube in place and with a diagnosis of herniation of stomach through the left diaphragm. On introducing a Levine tube with suction 1,200 c.c. of dark fluid and much gas was obtained. The heart shifted back to the midline, respirations and pulse rate decreased. X-ray examination showed a marked decrease

in the size of the stomach and the heart again in normal position (Fig. 13). Chemotherapy was started and the patient prepared for operation. He was operated upon under intratracheal anesthesia. The fractured eighth rib was widely resected. The omentum was found adherent in the wound of entrance in the chest wall. The pleural cavity was then entered.

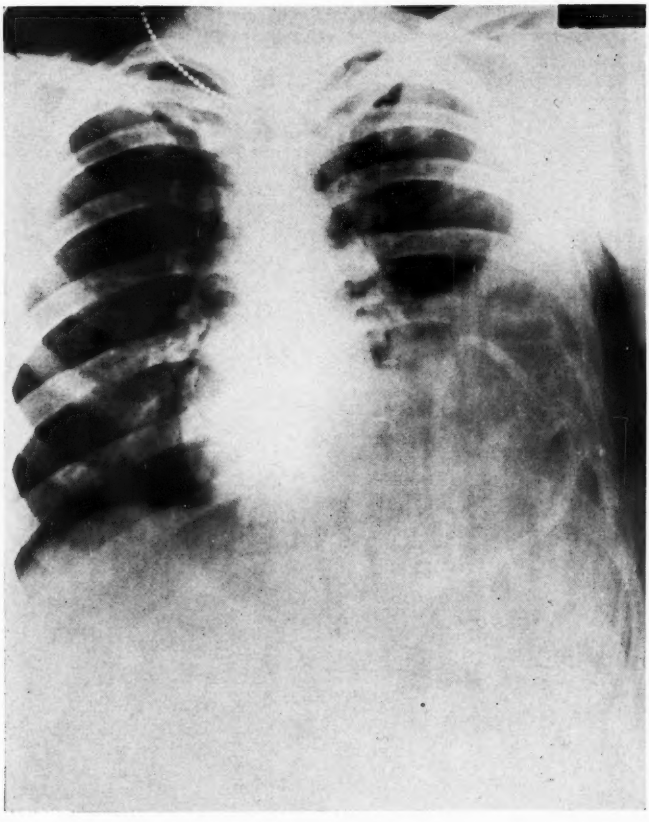

Fig. 13 (J. C.).—Levine tube passed. Stomach emptied. Heart and mediastinum have shifted back to midline. Patient immediately recovered from shock condition and prepared for operation.

The entire stomach and a portion of the transverse colon and omentum were in the left pleural cavity and had twisted 180 degrees through the longitudinal axis of the stomach. The organs were adherent to the torn edges of the diaphragm. They were separated. The diaphragm was incised for about 6 cm. to permit proper reduction of the hernial contents into the peritoneal cavity with exploration. The phrenic nerve was crushed. The wound of the diaphragm was debrided. The hernial aperture was repaired by interrupted mattress sutures of silk reinforced by fine sutures of silk. The chest was closed with air-tight underwater drainage and the air sucked out of the pleural cavity. The patient had to be evacuated to a holding company and was later evacuated to the zone of the interior.

The patient now lives in Philadelphia and through the courtesy of Dr. Gilson Engel and Dr. Farrell, this patient has been examined by fluoroscopy in December, 1947. Everything has been found normal with the diaphragm well healed.

CASE 3.—A sergeant was admitted in shock following a jeep accident. The right chest was crushed and there was a fracture of the right femur. He was treated for shock with blood and plasma. On admission his blood pressure was 60/40 with pulse 136. Penicillin and sulfonamide therapy were started. When the patient's condition was satisfactory, he was operated upon under intratracheal anesthesia. A transverse incision was made from the costosternal junction on the right side to the posterior axillary line over the sixth, seventh, and eighth ribs. These ribs were severely comminuted and the parietal pleura badly torn. The pleural cavity was filled with about 600 c.c. of bright red blood mixed with fragments of liver tissue. The lung was contused but intact. There were two lacerations of the diaphragm, probably caused by the rough comminuted rib ends. These two lacerations were joined exposing a large wound of the dome of the right lobe of the liver which was bleeding and many loose fragments of liver tissue were in this hole and in the pleural cavity. A subcostal incision was then made through the abdomen, and two gauze packs and a Penrose drain were placed into the large hole in the liver measuring about 10 cm. in diameter. These were brought out through the abdominal incision. The diaphragm was then sutured with interrupted silk mattress sutures reinforced with chromic sutures. The pleural cavity was then irrigated with saline and the chest wall closed in layers. The pleura could not be sutured as it was too badly torn. The lung was inflated, air sucked out of the pleural cavity, and air-tight underwater drainage instituted in the eighth interspace posteriorly.

The fractured femur was in a Thomas splint and temporarily left in it. The pleural drain was removed on the third postoperative day and a cast applied to the fractured femur. On the seventh day the liver tampons were removed and the Penrose drain left in situ. The chest wound was healed by primary union. The patient was evacuated to the zone of the interior.

Since that time, we have been able to communicate with this patient in February, 1948, and found out that the Penrose drain was removed at a rear installation immediately upon arrival. He then developed a subphrenic abscess which perforated through the torn diaphragm into the pleural cavity giving him an empyema. This was drained and healed without further complication, and he is at present perfectly well.

COMMENT

1. An understanding of the normal anatomy and the embryologic development of the diaphragm is necessary to properly evaluate the surgical problems presented.

2. Harrington and Marks have both given us excellent workable classifications of diaphragmatic hernias.

3. Certain types of hernias are amenable to surgical repair while others cannot be repaired.

4. The true short esophagus with thoracic stomach cannot be treated surgically. It is a rare anomaly. All or nearly all of the stomach is in the mediastinum.

5. Diaphragmatic hernia with shortened esophagus may be treated surgically by permanent phrenic section, freeing of the esophagus for lengthening, and closing the diaphragm above the cardia. Constriction at the junction of the esophagus with the stomach may cause dysphagia in these cases.

6. Hiatus hernias are the most common type. The esophagus is not short. It can be repaired surgically if the symptoms warrant it. The thoracic approach is probably the best and most practical, although this may be a matter of choice with the individual surgeon.

7. Congenital hernias occur most commonly through the foramen of Bochdalek, through the absence of a portion of the diaphragm; least frequently through the foramen of Morgagni, which hernias usually develop later in life. The earlier in life congenital hernias are operated upon the better. There is no benefit in delay. Two-stage closure of the abdomen in some instances will lower the mortality. The abdominal approach is by far the best. Circulatory, respiratory, and gastrointestinal symptoms are severe and may cause death by suffocation and cardiovascular collapse. In hernias through the foramen of Morgagni the abdominal approach should also be employed.

8. Eventration of the diaphragm is an infrequent condition. Symptoms are less marked than in hernias. The main differential diagnosis is between it and a true congenital hernia. Surgery is not indicated.

9. Congenital absence of the diaphragm is rare. It must be proved by surgical exploration but must previously be considered in the differential diagnosis.

10. Traumatic hernias are accompanied by severe symptoms, often with signs of intestinal obstruction. They interfere with the circulatory and respiratory symptoms. Surgery is indicated. The transthoracic approach is best.

CONCLUSIONS

1. Diaphragmatic hernia is diagnosed much more frequently now than formerly.

2. It must always be considered in the differential diagnosis of upper abdominal pathology.

3. Surgery will repair many of these hernias successfully.

4. Intratracheal anesthesia with positive pressure should be available.

5. Proper preoperative preparation, meticulous operative technique, and careful postoperative care will lower the former high mortality.

6. All types of diaphragmatic hernias are discussed and their treatment of choice enumerated.

7. Several case reports are included.

REFERENCES

1. Äkerlund, A.: Die Anatomische Grundlage des Röntgenbildes der sogen. "erworbenen Hiatus-brüche," Acta radiol. **14**: 523-544, 1933.
2. Bailey, P.: Case of Thoracic Stomach, Anat. Rec. **17**: 107, 1919.
3. Beckman, E. H.: Diaphragmatic Hernia With Report of 3 Cases, Surg., Gynec. & Obst. **9**: 154-156, 1909.
4. Bowditch, H. I.: Treatise on Diaphragmatic Hernia, Buffalo, 1853, Jewett, Thomas & Company.
5. Bowen, A.: Diaphragmatic Hernia—Review of the Literature, Am. J. Surg. **39**: 4-11, 1938.
6. Bright: Account of a Remarkable Misplacement of the Stomach, Guy's Hosp. Rep. **1**: 593-603, 1836.
7. Bund, R.: Ein Fall von rechtseitiger Hernia diaphramatica mit Austritt des Magens in den persistierenden Rezessus pneumatoentericus dextra, Frankfurt. Ztschr. f. Path. **21**: 243-257, 1918.
8. Donovan, E. J.: Congenital Diaphragmatic Hernia, Ann. Surg. **108**: 374-388, 1938.
9. Donovan, E. J.: Congenital Diaphragmatic Hernia, Ann. Surg. **122**: 569-581, 1945.
10. Dorsey, J. M.: Principles Involved in the Surgical Treatment of Diaphragmatic Hernias in Children, Am. J. Surg. **12**: 267-274, 1942.

11. Giffin, H. Z.: Diagnosis of Diaphragmatic Hernia, Ann. Surg. **55**: 389-399, 1912.
12. Harrington, S. W.: Diaphragmatic Hernia, Symptoms and Surgical Treatment in 60 Cases, J. A. M. A. **101**: 987-994, 1933.
13. Harrington, S. W.: Diaphragmatic Hernia: Results of Surgical Treatment in 210 Cases, California & West. Med. **51**: 27, 1939.
14. Harrington, S. W.: Diagnosis and Treatment of Various Types of Diaphragmatic Hernia, Am. J. Surg. **50**: 381-446, 1940.
15. Harrington, S. W.: The Surgical Treatment of the More Common Types of Diaphragmatic Hernia, Ann. Surg. **122**: 546-568, 1945.
16. Harrington, S. W.: Diaphragmatic Hernia, in Lewis's Practice of Surgery Vol. 5, Hagerstown, Md., 1948, W. F. Prior Company, Inc. Chap. 7, pp. 1-62.
Harrington, S. W., and Olsen, A. M.: Esophageal Hiatal Hernias of the Short Esophagus Type: Etiologic and Therapeutic Considerations, J. THORACIC SURG. **17**: 189-209, 1948.
17. Harris, W., and Clayton-Greene, W.: Congenital Absence Left Half of Diaphragm, Simulating Pneumothorax, Proc. Roy. Soc. Med. **5**: 153-156, 1911-12.
18. Hedblom, C. A.: Diaphragmatic Hernia—a Study of 378 Cases in Which Operation Was Performed, J. A. M. A. **85**: 947-953, 1925.
19. Hedblom, C. A.: Diaphragmatic Hernia, Ann. Int. Med. **8**: 156-176, 1934.
20. Ingals, E. F.: Diagnosis of Diaphragmatic Hernia, J. A. M. A. **36**: 1770-1774, 1901.
21. Jenkinson, E. L.: Absence of Left Half of Diaphragm (Thoracic Stomach Diaphragmatic Hernia), Am. J. Roentgenol. **26**: 899-903, 1931.
22. Jones, C. M.: Hiatus Esophageal Hernia: With Special Reference to Comparison of Its Symptoms With Those of Angina Pectoris, New England J. Med. **225**: 963-972, 1941.
23. Korns, H. M.: Diagnosis of "Eventration" of Diaphragm, Arch. Int. Med. **28**: 192, 1921.
24. Ladd, W. E.: Congenital Absence of Pericardium. Report of a Case, New England J. Med. **214**: 183-187, 1936.
25. Ladd, W. E., and Gross, R. E.: Congenital Diaphragmatic Hernia. New England J. Med. **223**: 917-925, 1940.
26. LeWald, L. T.: Thoracic Stomach, Differentiation From Eventration and Hernia of Diaphragm, Radiology **3**: 91-104, 1924.
27. LeWald, L. T.: Congenital Absence of Left Half of Diaphragm, Arch. Surg. **14**: 332-344, 1927.
28. Lord, F. T.: Eventration of the Diaphragm, Arch. Surg. **14**: 316-329, 1927.
29. Marks, J. H.: Diaphragmatic Hernia and Associated Conditions, Am. J. Roentgenol. **37**: 613, 1937.
30. Orr, T. G., and Neff, F. C.: Diaphragmatic Hernia in Infants Under One Year of Age Treated by Operation, J. THORACIC SURG. **5**: 434-440, 1936.
31. Rives, J. D., and Baker, D. D.: Anatomy of the Attachments of the Diaphragm: Their Relation to the Problems of the Surgery of Diaphragmatic Hernia, Ann. Surg. **115**: 745-755, 1942.
32. Sagal, Z.: Absence of Left Diaphragm Associated With Inverted Thoracic Stomach, Am. J. Roentgenol. **30**: 206-214, 1933.
33. Sweet, R. H.: The Repair of Hiatus Hernia of the Diaphragm by the Supra-diaphragmatic Approach, New England J. Med. **238**: 649-653, 1948.
34. Truesdale, P. E.: Diaphragmatic Hernia at the Esophageal Hiatus, Short Esophagus and Thoracic Stomach, New England J. Med. **212**: 240-250, 1935.
35. Truesdale, P. E., and Phippen, W. G.: Traumatic Diaphragmatic Hernia Following War Injuries, New England J. Med. **212**: 697-702, 1935.
36. Truesdale, P. E.: Diaphragmatic Hernia in Children With a Report of 13 Operative Cases, New England J. Med. **213**: 1159-1172, 1935.
37. Weintraub, S., and Tuggle, A.: Duodenal Diverticula, Radiology **36**: 297-301, 1941.

LIPOID PNEUMONIA RESULTING FROM A DERMOID CYST

Case Report

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THE case presented herewith is deemed worthy of reporting because of the unusual source of the lipoid material which produced the lipoid pneumonia.

Lipoid pneumonia is customarily considered to have its origin from the inadvertent entrance of lipoid material into the tracheobronchial tree.

Animal and mineral oils are known to cause tissue reactions, whereas neutral vegetable oils have been shown to be practically innocuous.[1, 2, 3]

The diagnosis of lipoid pneumonia in the case herewith presented was first established by a biopsy taken November, 1945, two years before the patient's present entry, when a mediastinal lymph node showed scarring and high fat content. It was again confirmed by us bronchoscopically, at the present entry. Operation revealed the unexpected finding of a large dermoid cyst, the cavity of which contained viscid animal oil. This cavity communicated directly into the primary bronchus of the left upper lobe. The lipoid substance was thus disseminated directly into the left upper lobe and by spill-over to a lesser extent, into the lower lobe. This source accounted for the unilateral and diffuse character of the lesion which involved all of the left lung but with maximum disease in the upper lobe into which the dermoid directly drained. The history of the use of oily nose drops for many years by this patient suggested preoperatively this to be the source of her lipoid pneumonia. However, histochemical studies* of the oil in the lung after its extirpation have shown the offending oil to be the unsaturated type, thus excluding mineral oils. Therefore, we must conclude that the oil which caused the lipoid pneumonia was derived from the patient herself (i.e., the dermoid) and that the oily nose drops were merely incidental and had no bearing on the production of the lipoid pathology.

CASE REPORT

History (Essential).—The patient (D. P.) a 20-year-old white woman, well developed and fairly well nourished was first seen at the Mount Zion Hospital on July 28, 1944. Complaints then consisted of repeated attacks of cough, fever, and expectoration for as long as she could remember. She had used oily nose drops frequently and regularly since childhood. At this time there were also painful swellings of the joints, especially of the fingers and left wrist. These were suggestive of rheumatoid arthritis. Routine blood and urine studies were negative except for an elevated sedimentation rate. X-ray examination of the chest showed a 10 by 5 cm. hilar mass extending towards the left, suggestive of tuberculosis or lymphoma. A therapeutic trail of x-ray radiation was given with no resolution of the hilar mass.

During the subsequent fourteen months she was readmitted to the hospital three times with the same complaints. Bronchoscopy then revealed what was considered to be a mild

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*The histochemical analyses were made in the Department of Pathology of Mount Zion Hospital, by Dr. Gerson R. Biskind, Chief of the Department.

tracheobronchitis. Bronchograms showed the anterior and middle bronchi of the left upper lobe to be spread apart, apparently by extrinsic pressure resulting from a mass in the anterior mediastinum.

The mediastinal mass continued to enlarge and operative intervention was undertaken* in October, 1945. A dense mass of mediastinal nodes was encountered which, in spite of their previous failure to respond to radiation therapy, was considered to be lymphomatous in nature. Specimens were removed for histologic study and the wound was closed. Microscopic examination of these removed specimens was reported as showing hyperplasia of the regional lymph nodes with marked deposition of lipoid material in the sinusoids. The diagnosis was then considered to be lipoid pneumonia with regional lymph node involvement, the causative factor being attributed to the persistent use of oily nose drops.

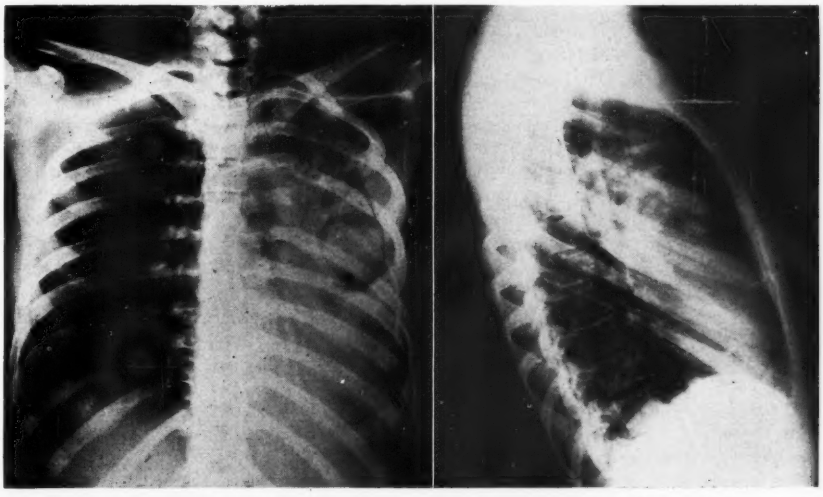

Fig. 1.—X-ray examination anteroposterior and lateral views. Shift of trachea and mediastinum to the left; entire left chest full of areas of varying degrees of increased density with peculiar rounded shadows of increased density currently surrounded by air in the midperipheral portion; thickened pleura; regenerated fifth rib.

Cessation in the use of the oily nose drops did not improve matters. Cough and bouts of fever increased in frequency, the sputum became very copious and finally foul; there ensued mild and severe hemoptyses. The mediastinal mass grew and both lobes of the left lung became involved in a chronic pneumonic process, most marked in the left upper lobe. Laboratory studies except for the continued elevated sedimentation rate remained essentially normal. These included negative tuberculin up to 1:100 and sputum studies for tumor cells.

The patient was first seen by me late in 1947, when, in view of the preceding course and findings, it was decided that nothing short of extirpation of the involved tissues might be of benefit. The lesion was considered to be a diffuse lipoid pneumonia, with marked bronchiectatic changes and extensive mediastinal lymph node involvement.

OPERATION

Dec. 30, 1947, preoperative diagnosis: paraffinoma left lung. Operation: left pneumonectomy. Anesthesia: intratracheal.

Procedure.—The chest cavity was opened through the fifth intercostal space. There was no free pleural space. The lung was adherent to the chest wall on all sides and

*By Dr. Clayton Lyons.

moderately so to the pericardium. With blunt and sharp dissection the lung was freed from the chest wall. The large adhesions were clamped and tied. The upper and lower lobes of the lung had the consistency of liver tissue. They did not collapse nor did they fully expand. The upper lobe was a little more involved than the lower lobe but both were definitely fibrotic. The left leaf of the diaphragm did not move too well on respiration. The lateral half did not seem to move at all.

Lying in the anterior mediastinum just under the sternum and in close relation to the root of the lung was a mass about the size of a turkey egg which at first was thought to be a lymph gland but when it was accidentally opened, it was found to be a dermoid cyst containing hair and an oily cheeselike material. After fine dissection the pulmonary artery was isolated and divided between ligatures. The inferior pulmonary vein was isolated; it coursed for a distance of 2 cm. and then divided into two main branches. The vein was then cut between ligatures. A similar procedure was carried out on the superior left pulmonary vein. The left main bronchus was then isolated and two ligatures of heavy white braided silk ties were placed and the bronchus was cut through. The lung was then removed and the bronchial stump was closed with 1-No. 1 silk interrupted sutures. The chest cavity was filled with normal saline and no air was seen to bubble through so that the bronchial stump was completely closed. All of the saline was aspirated by suction. No gross bleeding was evident. The closure of the chest cavity was undertaken. Sterile dry dressings were applied and the patient was turned on her back and bronchoscopy was done. The main bronchus to the right lung was found to be clear and there was considerable mucus in the posterior nasal pharynx which was aspirated and the patient was returned to the ward. During the procedure the patient received about two and one-half units of whole blood with the completion of the third taking place on the ward.

Postoperative diagnosis: (1) dermoid cyst anterior mediastinum; (2) paraffinoma left lung.

PATHOLOGY

Gross Description.—The specimen consisted of a left lung, a rounded mass of yellow-white tissue, and several smaller rounded masses of tissue said to represent hilar lymph nodes. The lymph node masses varied in size up to 3 cm. in diameter. They were firm and on section had a cream-yellow parenchyma which was mottled by red-brown zones. Several smaller similar lymph nodes were attached to the hilar portion of the lung. The lung possessed two lobes. The line of resection was 1 cm. proximal to the first division of the left main bronchus. The lung was 16 cm. from apex to base, 11 cm. wide, and 5 cm. in thickness. The pleura was red-brown in color and roughened by numerous thin tags of fibrous tissue. Near the center of the left upper lobe there was a space which was irregularly rounded and slightly collapsed. It had a diameter of 5 cm. This cavity lay 2.5 cm. from the interlobar septum. It was covered by 0.7 cm. of lung tissue on its medial aspect. The inner surface of this cavity was shaggy with a gray-brown color. It contained yellow cheesy material and numerous blond hairs. A communication from this cavity into a large branch of the upper lobe bronchus was identified. The rounded yellow-cream-colored mass mentioned previously was said to have occupied the cavity within the lung parenchyma and to have been freed from this space during surgery. This mass measured 7 cm. in greatest length and 5 cm. in greatest width. It was irregularly rounded. Its surface had the appearance of skin and bore numerous blond hairs. It was firm and on section it was composed of yellow tissue containing numerous streaks of white tissue. In one zone the mass contained a cyst 2 cm. in diameter. The cyst was filled with cheesy material and pale hairs. One pole of this mass was covered by a sheet of gray-brown and fatty tissue varying in thickness from 0.3 to 1 cm. This tissue was said to have been removed from the mediastinum. The mass was attached to this encapsulating tissue. The parenchyma of the upper lobe was extremely solid. It had a mottling of yellow, cream, tan, and brown areas. There was accentuation of the interlobular septa. The pulmonary tissue was atelectatic in many places. The lower lobe was poorly aerated, though it was not as dense as the upper lobe. The lobular markings were not as prominent. The parenchyma varied from tan to brown.

Microscopic Description.—The rounded mass which filled the cavity in the lung was composed of dense fibrous tissue covered on one aspect by cornified stratified squamous epithelium. In the fibrous tissue were nests of adipose tissue, and under the epithelium were hair follicles and numerous large sebaceous glands. Lymphocytes, plasma cells, macrophages, and a few polymorphonuclear cells were scattered throughout the fibrous stroma. The cystic portion of the mass was lined also by cornified stratified squamous epithelium. Sebaceous glands were also present. In addition, there was a large group of glands in a lobular arrangement around the cyst. These glands consisted of dark and light-staining columnar cells arranged in acinar groups, that communicated with tubular structures lined by a single regular layer of tall pale cells. This zone represented serous and mucous salivary gland elements.

Fig. 2.—Gross section after pneumonectomy showing: Grumous material of the dermoid on the mediastinal surface of the lung and the more solid material and hair which was removed from the cavity in which the dermoid was developing.

The lung tissue which lined the cavity had a layer of granulation tissue on the inner surface. The granulation tissue consisted of young fibrous tissue, numerous small blood vessels, lymphocytes, plasma cells, macrophages, giant cells, and polymorphonuclear cells. Fragments of stratified squamous epithelium were scattered on the surfaces of the granulation tissue. The lung parenchyma showed profound changes; no normal lung tissue remained in this zone. There was severe and extensive fibrosis of the interstitial and interlobular tissue; there was severe distortion of alveoli with dense infiltrates of lymphocytes, plasma cells, macrophages, and giant cells in the interstitial tissue and in the alveolar walls. Polymorphonuclear cells were also present in some areas. The macrophages were full of fatlike material; macrophages commonly filled alveoli. Some of the giant cells had vacuoles and clefts within their cytoplasm. In regions more distant from the site of the tumor the alveoli contained blood and serum, and the bronchi contained a polymorphonuclear exudate. The interstitial tissue in

general was increased, particularly in the interlobar and interlobular septa. The changes in the lower lobe varied in intensity, generally the interstitial scarring was present and the inflammatory reaction in the alveoli was represented by blood serum and macrophages.

Fig. 3.—Gross cut section of the lung showing the pressure defect in the upper lobe simulating a cavity made by the dermoid with a probe passing from the cavity directly into the left upper lobe bronchus.

A section that represented the bronchus that was traced into the cavity showed a dense chronic inflammatory infiltrate in the bronchial mucosa and surrounding interstitial tissue. The columnar epithelium of the bronchus changed into stratified squamous epithelium; beneath this epithelium were sebaceous glands.

The hilar and mediastinal lymph nodes were enlarged. They had prominent follicles with distinct germinal centers. The sinusoids were lined by many layers of endothelial cells. The sinusoids contained lymphocytes, neutrophiles, eosinophiles, macrophages, and some giant cells. Large and small vacuoles were scattered throughout sinusoids of the lymph nodes.

Fat stains were prepared of the lung and lymph node. With the Scharlach R stain, red droplets were present in the pulmonary parenchyma in the region of the tumor. The fat droplets were very small in the macrophages, in the alveoli, and in the columnar epithelial cells of the bronchi of that region. The fat droplets were larger in the interstitial tissue where they may be engulfed by giant cells or free in the fibrous elements. Here they had the appearance noted in the original biopsy.

There was less fat in the interstitial tissue of the lower lobe and practically none in the alveoli of that lobe. The same blocks of tissue were also stained with osmic acid and the preparations are compared with the Scharlach R stain. It was noted that the droplets that stained red with Scharlach R were also stained black with osmic acid. There was no apparent difference in the quantity of fat demonstrated by the two techniques. Fat droplets were demonstrated in the lymph nodes by these two techniques.

DIAGNOSIS*

1. Dermoid tumor of mediastinum, situated in upper lobe of left lung, communicating with major bronchus.
2. Chronic patchy organizing pneumonitis, left lung, with interstitial scarring due to lipoid from dermoid tumor.
3. Reactive hyperplasia of regional lymph nodes with lipoid in sinusoids.

REFERENCES

1. Brown, A. Lincoln, and Biskind, G. R.: Differential Diagnosis Between Lipid Pneumonia and Pulmonary Neoplasm, J. A. M. A. **117**: 4-6, 1941.
2. Pinkerton, Henry: Fats and Oils: Their Entrance Into and Fate in Lungs of Infants and Children: Clinical and Pathologic Report, Am. J. Dis. Child. **33**: 259, 1927; Reaction to Oils and Fats in Lung, Arch. Path. **5**: 380, 1928.
3. Janes, Robert M.: Lipoid Pneumonia Simulating Bronchiogenic Carcinoma, J. THORACIC SURG. **16**: 451-457, 1947.

* Gerson R. Biskind, Pathologist.

ACUTE PUTRID ABSCESS OF THE LUNG

VI. The Late Results of Surgical Treatment

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DURING the decade prior to World War II, members of our Thoracic Group at The Mount Sinai Hospital published a series of papers concerning various aspects of putrid pulmonary abscess.[1-18] We, among the participants, described the technique of one-stage drainage in the acute phase of the disease and reported the results derived therefrom.[7, 8, 9, 10, 12] At the time, we summarized our experiences by stating that (1) "cure is achieved without hazard in the great preponderance of cases," and (2) "the results are excellent, not only as to recovery from operation but also as to complete and early restoration of health." The foregoing statements were based upon follow-up observations which, at the time, varied from 3½ months to 10 years in duration.

The passing years have afforded us an opportunity to observe many of our patients for much longer periods; and it is now our purpose to set forth the late results of operation in order to demonstrate that they are not quite as satisfactory as we had assumed originally. This will be done by reviewing briefly the cases of acute putrid pulmonary abscess that we subjected to one-stage drainage prior to Jan. 1, 1945. Up to that date, we had a total of 165 such cases but we shall confine our analysis only to those that could be followed for at least 5 years after operation. It is our opinion that if one adheres to certain strict criteria of "cure," such a long period of follow-up is not essential in order to make certain that the patient will remain well. However, since this communication deals with "late results," we have arbitrarily selected 5 years as the minimum period of observation of all cases to be included in our report.

Among our 165 patients, 154 were operated upon in the "pre-penicillin era." There were 4 operative deaths in our series. The latter, constituting an operative mortality rate of 2.43 per cent, have been reported elsewhere.[12] Of the 161 patients who survived operation, 46 could not be followed for the required minimum period of 5 years and therefore have been eliminated from further consideration. (Of these 46 patients, 4 are known to be dead. One died following an operation for strangulated hernia and 2 died of malignant disease of the pancreas and colon, respectively. The fourth patient died of lobar pneumonia more than a year after the operation for pulmonary abscess. All 4 patients were free of symptoms of pulmonary abscess at the time of death and, in each instance, the latter was due to a cause unrelated to the original pulmonary infection. The remaining 42 patients gradually disappeared from observation. When last seen all were free of symptoms, some having been followed for more

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than 4 years after operation.) After eliminating the 4 operative deaths and the 46 cases that could not be followed for the required minimum period of 5 years, 115 cases remained. These cases, which were observed for 5 to 25 years, form the basis for certain conclusions that will be presented at another point. We have divided the cases into two groups, according to certain strict criteria.

Group A consists of patients whom we consider "cured." In order to be classified under Group A, the patient must (1) be entirely symptom-free, (2) have a healed wound, and (3) have a normal chest film or bronchogram. There were 100 patients (86.9 per cent of the 115 cases) in this group. Four have been followed for 20 to 25 years, 7 for 15 to 20 years, 12 for 10 to 15 years, and 77 were under observation for 5 to 10 years.

Group B is comprised of those patients who, in the light of the criteria of "cure" enumerated in the preceding paragraph, cannot be considered "cured" despite the fact that most of them became free of symptoms of pulmonary abscess soon after operation. In this group, there were 15 patients (13.04 per cent of the 115 cases). On x-ray examination, all presented either a small residual pulmonary cavity or persistent pulmonary infiltration. On bronchography, varying degrees of bronchial dilatation were noted in all, but it is to be emphasized that *none* of them presented symptoms of residual pulmonary suppuration. *Nine became entirely symptom-free shortly after operation and have remained so.* The remaining 6 patients either have, or have had in the past, recurring episodes of hemoptysis. One patient, who has been entirely free of symptoms since operation, has had a small persistent bronchocutaneous fistula.

The 15 patients in Group B serve to re-emphasize a viewpoint that we expressed many years ago, namely, that *regardless of absence of evidence of pulmonary suppuration a patient who has had a pulmonary abscess cannot be considered well unless the x-ray film discloses complete disappearance of pulmonary infiltration.*[17] As mentioned already, 6 of our 15 patients have suffered recurrent small hemoptyses. These cases have been observed for 7 to 16 years. It is important to note that while 3 patients developed hemoptysis shortly after operation, the remaining 3 *did not begin to bleed until some years after operation.*

Careful analysis of the histories of the 6 patients, who developed hemoptysis, disclosed that one bled occasionally during the 7 years that we were able to follow him after operation. The second patient has had identical manifestations for 16 years. The third patient had occasional hemoptysis for the first 8 years and has remained well for the past 6 years. The fourth patient was entirely free of symptoms for 4 years and has bled, from time to time, during the past 3 years. The fifth patient was well for 5 years, then had recurrent hemoptysis for 2 years and has been well for the past year. *The last patient was well for 10 years but, during the last 4 years, has had recurrent small hemoptyses.*

Of the 9 patients who remained symptom-free following operation despite the fact that their x-ray films were abnormal, 1 has been followed for 7 years, 2 for 8 years, 2 for 9 years, 2 for 14 years, 1 for 21 years, and the last for 22 years. In view of the fact that certain of the patients mentioned in the pre-

ceding paragraph did not commence to bleed for a number of years after operation, there is no assurance that these 9 patients will not have hemoptysis in the future. Bronchography has been performed in all 9 cases and, in each instance, demonstrates the presence of bronchial dilatation of varying degree and extent. In view of the already-demonstrated tendency of such patients to bleed, *a guarded prognosis must be given in all* regardless of the fact that all of them have remained well for 7 years or longer.

With the object of attempting to determine the factors responsible for the persistence of pulmonary cavities or bronchiectasis following drainage of an acute putrid pulmonary abscess, the 15 cases of Group B were reviewed. In each instance, operation was performed within 10 days to 5 weeks of the onset of symptoms; and, accordingly, chronicity of the pulmonary abscess can be excluded as a cause of the residual pulmonary abnormality. The first apparently significant finding was that in 8 patients the abscess cavity, at operation, was noted to be three inches or more in diameter. In 2 of these cases, profuse hemoptysis occurred prior to operation; and, at operation, the cavity was found to measure more than four inches in diameter. In practically all patients with large cavities, the clinical course was characterized by high fever, prostration, and apathy or other evidence of severe toxemia. In 3 cases which presented such clinical features, the preoperative film disclosed very extensive pulmonary infiltration but, at operation, the pulmonary abscess did not exceed two inches in diameter. In another 2 cases in which dense shadows were noted in the preoperative roentgenogram, at operation the lesion was found to have already perforated into the pleura. In these 2 instances, several revisions of the operative wound and prolonged drainage were required. In only 1 case of the 15 was the cavity small (three-fourth inch in diameter) and the preoperative manifestations mild. This patient has remained symptom-free for 7 years since operation, but the postoperative bronchogram revealed varicose dilatation of the branches of the left lower lobe bronchi in the vicinity of the original pulmonary abscess. From all of the foregoing, it appears that cases characterized preoperatively by extensive pulmonary excavation, profuse hemoptysis, and severe symptoms are likely to be complicated postoperatively by persistence of pulmonary cavitation or bronchiectasis, or both. These may occur regardless of the fact that operation is performed at a relatively early stage of the disease.

From a practical standpoint, the question arises whether the cases which we have just discussed might have been treated more effectively by some method other than that employed. In this connection, it is to be emphasized that all but 11 of our patients were treated prior to the time that penicillin and other antibiotics became available to us. Without burdening the reader with a mass of statistics, it may be stated that many a patient with a large acute pulmonary abscess who presented a fulminating preoperative clinical course (hyperacute abscess) made an uneventful convalescence and has remained entirely well for many years following simple surgical drainage.[12] On the other hand, we have presented evidence to indicate that other cases of the same general type, although relieved of symptoms of pulmonary suppuration after operative drain-

age, may be left with pulmonary cavities or bronchiectatic dilatations which later are a source of hemoptysis. As regards primary lobectomy or segmental resection, we are unaware of any reports in the literature concerning such treatment in a large series of cases of "hyperacute" putrid pulmonary abscess, during the period in which we treated such cases by "one-stage drainage." Accordingly, we are unable to state the mortality, morbidity, and incidence of satisfactory results that might have followed pulmonary resection under the circumstances outlined previously.

Among 154 cases of acute putrid pulmonary abscess operated upon by us in "the pre-penicillin era" (prior to January, 1943), 41 were of the "hyperacute" variety. Three of our 4 operative deaths occurred in this group.[12] Thus, simple drainage of these fulminating lesions resulted in a mortality rate of 7.3 per cent prior to the advent of modern antibiotic therapy. All 41 patients suffering from "hyperacute" abscess were so ill at the time of operation, that it seems unlikely that they could have withstood any procedure other than a rapidly performed, emergency drainage under local anesthesia. Specifically, we maintain that lobectomy or segmental resection would have resulted in greater morbidity or mortality than that which resulted from drainage.

Since the advent of antibiotics, there has been a general tendency on the part of surgeons to treat chronic pulmonary abscess by means of resection rather than drainage. There also has been a parallel trend in the treatment of acute putrid abscess of the lung. Up to the present writing, however, we have not encountered any reports of combined pulmonary resection and antibiotic therapy in the treatment of a substantial number of cases of "hyperacute" abscess. Thus, the results of such therapy in "hyperacute" cases still remain in doubt although the results in less severe cases appear to be excellent, not only as regards low morbidity and mortality but also high incidence of cure. For the time being the following mode of treatment of fulminating cases of acute putrid pulmonary abscess appears most logical:

a. In cases in which intensive antibiotic therapy does not result in prompt fall of temperature, reduction of the amount of sputum, and diminution of toxic manifestations, the safest procedure appears to be one-stage drainage. Should the pulmonary cavitation or bronchiectasis persist, secondary lobectomy or segmental resection should be performed at a later date, namely, when the element of gross infection has been eliminated.

b. In cases in which antibiotic therapy results in prompt control of infection, primary lobectomy or segmental resection appear to be the methods of choice.

SUMMARY AND CONCLUSIONS

Prior to Jan. 1, 1945, the authors subjected 165 patients suffering from acute putrid pulmonary abscess to operation; 154 of these cases were operated on in the "pre-penicillin" era.

Operation consisted of drainage in one stage, according to a technique described previously.

Among the 165 cases, there were 4 operative deaths (an operative mortality rate of 2.43 per cent).

Of the 161 survivors, 46 could not be followed for a minimum period of 5 years and therefore were eliminated from further consideration.

One hundred fifteen cases, followed for from 5 to 25 years, form the basis of this report.

The 115 cases were divided into two groups, according to strict criteria.

Group A consists of patients who are considered "cured." They are free of symptoms, have healed wounds, and have negative chest films or bronchograms. There are 100 patients in this group (86.9 per cent of 115 cases).

Group B consists of patients who cannot be considered well because of the fact that they have *residual postoperative pulmonary cavities or bronchiectasis.* There are 15 cases in this group (13.04 per cent of 115 cases). *None of them has symptoms of residual pulmonary suppuration. Nine have been entirely symptom-free.* The remaining 6 patients have had hemoptysis at one time or another. Because, in some cases, bleeding occurred quite a number of years after operation, it is conceivable that the 9 patients who have been free of hemoptysis may bleed in the future. Under any circumstances, 13.04 per cent of 115 cases of acute putrid pulmonary abscess cannot be considered "cured," following one-stage drainage. This is a higher incidence of unsatisfactory results than we had assumed previously on the basis of a shorter period of follow-up observation.

Practically all patients in Group B presented large abscesses, extensive areas of pulmonary infiltration, profuse hemoptysis, or severe clinical symptoms, prior to operation.

Among the 165 cases, 41 were of the above-mentioned "hyperacute" type. One-stage drainage in these 41 cases resulted in 3 operative deaths (an operative mortality rate of 7.3 per cent). However, the majority of patients suffering from "hyperacute" abscess responded well to the one-stage drainage procedure.

While primary lobectomy or segmental resection constitute theoretically ideal procedures in cases of "hyperacute" abscess, in so far as they would appear to eliminate the danger of residual pulmonary cavitation and bronchiectasis, it seems fair to assume that such radical surgery would have been attended by a significantly higher morbidity and mortality than followed the one-stage drainage operation before the advent of antibiotics.

Since the advent of antibiotics, the ideal appears more capable of being achieved.

REFERENCES

1. Antopol, W., and Rabin, C. B.: Purulent Thrombophlebitis of the Pulmonary Vein, Arch. Path. **16:** 435, 1933.
2. Cohen, J.: Bacteriology of Abscess of the Lung and Methods for Its Study, Arch. Surg. **24:** 171, 1932.
3. Glass, A.: The Bronchopulmonary Segment With Special Reference to Putrid Lung Abscess, Am. J. Roentgenol. **31:** 328, 1934.
4. Kramer, R., and Glass, A.: Bronchoscopic Localization of Lung Abscess, Ann. Otol., Rhin. & Laryng. **41:** 1210, 1932.
5. Neuhof, H., and Wessler, H.: Putrid Lung Abscess, J. THORACIC SURG. **1:** 637, 1933.
6. Neuhof, H., and Hirshfeld, S.: Putrid Empyema, Ann. Surg. **100:** 1105, 1934.

7. Neuhof, H., and Touroff, A. S. W.: Acute Putrid Abscess of the Lung. I. Principles of Operative Treatment, Surg., Gynec. & Obst. **63**: 353, 1936.
8. Neuhof, H., and Touroff, A. S. W.: Acute Putrid Abscess of the Lung. II. An Analysis of Forty-five Consecutive Operative Cases, Surg., Gynec. & Obst. **66**: 836, 1938.
9. Neuhof, H., and Touroff, A. S. W.: Acute Putrid Abscess of the Lung. IV. Surgical Treatment and Results in Eighty-six Consecutive Cases, J. THORACIC SURG. **9**: 439, 1940.
10. Neuhof, H., and Touroff, A. S. W.: Acute Putrid Abscess of the Lung. A Surgical Disease, New York State J. Med. **40**: 849, 1940.
11. Neuhof, H., Touroff, A. S. W., and Aufses, H.: The Surgical Treatment, by Drainage, of Subacute and Chronic Putrid Abscess of the Lung, Ann. Surg. **113**: 209, 1941.
12. Neuhof, H., and Touroff, A. S. W.: Acute Putrid Abscess of the Lung. V. Hyperacute Variety, J. THORACIC SURG. **12**: 98, 1942.
13. Rabin, C. B., and Moolten, S. E.: Pyelephlebitis Complicating Gangrenous Pulmonary Abscess, Arch. Path. **16**: 436, 1933.
14. Stern, L.: Putrid Abscess of the Lung Following Dental Operations, J. THORACIC SURG. **4**: 547, 1935.
15. Touroff, A. S. W.: The Evacuation of Deep-Seated Abscesses, Ann. Surg. **94**: 477, 1931.
16. Touroff, A. S. W., and Moolten, S. E.: The Symptomatology of Putrid Abscess of the Lung, J. THORACIC SURG. **4**: 558, 1935.
17. Touroff, A. S. W., and Neuhof, H.: Acute Putrid Abscess of the Lung. III. Roentgenographic Features, Surg., Gynec. & Obst. **68**: 687, 1939.
18. Touroff, A. S. W., and Neuhof, H.: The Differentiation Between Acute Putrid and Nonputrid Pulmonary Abscess, J. THORACIC SURG. **10**: 618, 1941.

TRANSTHORACIC, THORACOABDOMINAL PORTACAVAL ANASTOMOSIS

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THE subject of portal cirrhosis has interested both internist and surgeon for many years. The prognosis in this condition has always been poor; death has been uncomfortably drawn out or has come with dramatic suddenness, accounted for by one or any combination of the following mechanisms:

A. Liver failure (cholemia)
B. Portal hypertension
   1. "Wasting ascites"
   2. Exsanguination, due to rupture of:
      (a) Esophageal varices
      (b) Gastric varices

The treatment of depressed liver function is medical. Remarkable improvement is generally obtained with a regime consisting of a high protein, high carbohydrate, low fat diet, lipotropic substances, supplementary vitamins and proteins, liver extract, etc. The problem of portal hypertension, on the other hand, is one of mechanics. Due to intrahepatic, periportal fibrosis, congestion in the portal system ensues. Thereafter, the increased intravenous hydrostatic pressure may cause ascites, or compensation may take the form of varicosities of the venous tributaries, particularly the esophageal and gastric network. More commonly ascites and varicosities coexist.

The methods devised to combat the disastrous sequelae of portal hypertension are numerous and ingenious. These include the Talma-Morison operation,[1, 2] insertion of the Crosby-Cooney button,[3] sclerosing of the esophageal varicosities,[4, 5] intraesophageal venous tamponage,[6] posterior mediastinotomy,[7] gastric resection,[8] and gastroesophagectomy.[9] Such methods, however, fail to strike at the critical point of obstruction, and are at best either partially or temporarily effective. The shunting of blood from the portal system directly into the systemic venous circulation is the most rational and effective method of dealing with portal hypertension. This method was proposed by von Eck in 1877,[10] and subsequently tried on human beings with varying and questionable success by Tansini (1902), Vidal (1903), De Martel and Lenoir (1910), Rosenstein (1911).[11] It remained, however, for the stimulating work of Whipple,[11, 12] and of Blakemore and Lord,[13, 14, 15] to renew interest in the concept of supplementing the collateral circulation between the portal and systemic venous systems, and to establish this therapy as one of choice in the management of portal hypertension.

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Portal hypertension may be due to an intra- or extrahepatic block. The incidence of intrahepatic block, notably portal cirrhosis, exceeds by far the relatively few cases of extrahepatic block which are generally due to thrombosis, scarring, or cavernomatous transformation of the portal or splenic veins.[16] In cases of extrahepatic block, a splenorenal shunt is indicated. In portal cirrhosis, however, either a splenorenal or a portal-inferior vena cava (Eck fistula) anastomosis is available. It is generally conceded that the Eck fistula procedure is more desirable.[12, 14, 17] In the first place, and most important, it is capable of shunting more blood than a splenorenal anastomosis. Normally, the splenic vein handles approximately 20 to 40 per cent of the total blood volume of the portal system. Splenorenal shunt, therefore, permits only a partial reduction in portal hypertension, generally between 100 and 150 mm. of water[14] and while such a drop may be sufficient in some cases to alleviate symptoms, closer approximation to the normal portal pressure of 100 mm. of water is desirable when dealing with abnormal pressures ranging between 350 and 600 mm. of water.

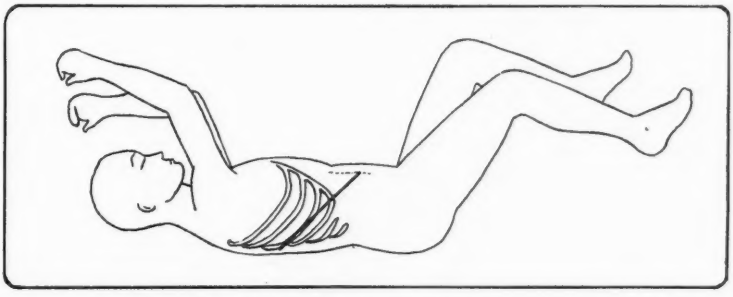

Fig. 1.—Position of patient and line of incision. The patient lies on the left side in the true lateral position. The incision is made directly over the ninth rib commencing at the lateral margin of the right erector spinae muscles and is continued onto the abdomen to the lateral border of the right rectus muscle.

A second objection to splenorenal shunt for portal cirrhosis is the greater likelihood of thrombus formation at the site of anastomosis. To offset this complication, heparin is generally employed postoperatively, a precaution beset with great hazard. Finally, splenorenal shunt is not as well tolerated by the patient as is an Eck fistula. The majority of cases of cirrhosis of the liver are associated with splenomegaly[18]; perisplenitis is the rule and the splenectomy which is a necessary preliminary to splenorenal shunt is accomplished with difficulty, if at all. A considerable number of cases of splenectomy in lieu of a proposed shunt operation never reach the literature, and fatal outcomes in these instances are listed as failures in splenectomy and not splenorenal shunts.

The objection of by-passing the liver with portacaval shunt (Eck fistula) on the grounds of possible nutritional difficulties[19] does not seem a valid one. McIndoe[20] demonstrated that in portal cirrhosis the maximum amount of blood reaching the liver via the portal vein is 13 per cent. The establishment of a splenorenal shunt will reduce even this amount to a negligible figure, even

though the portal vein itself is not molested. Moreover, Herrick[21] has shown by his perfusion studies that in the normal liver there is a 1 mm. rise in portal vein pressure for every 40 mm. rise in hepatic artery pressure, while in the cirrhotic liver the ratio is reduced to 1:6. Herrick further pointed out that an arterial rise in pressure does not affect the portal pressure in normal livers until the 100 mm. mark is passed, whereas in cirrhotic livers the influence is discernible with pressures as low as 30 mm. Such increased sensitivity of portal tension to hepatic artery alterations in pressure is another point in favor of portacaval as opposed to splenorenal shunt. It is, therefore, evident that the

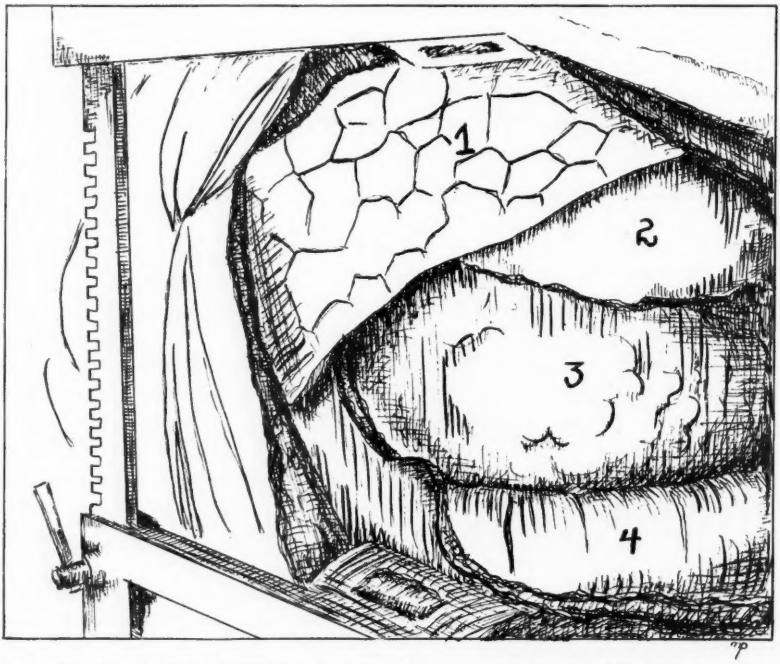

Fig. 2.—Exposure following resection of ninth rib: *1*, Lower lobe of right lung; *2*, upper leaf of incised diaphragm; *3*, liver; *4*, lower leaf of diaphragm.

Eck fistula offers the greatest possible relief of portal hypertension and should be employed in all but those cases in which other types of shunt operations are specifically indicated, such as the splenorenal[15] and combinations of the superior and inferior mesenteric with the vena cava, left ovarian, and left adrenal veins as reported by Linton.[22] In general, it would appear that the chief deterrent to the widespread adoption of the Eck fistula is the fact that when the usual abdominal approach is employed, it is a technically difficult procedure.

In an effort to simplify portal-inferior vena caval anastomosis, one of us (V. P. S.) has reported a transdiaphragmatic, thoracoabdominal approach.[23, 24] This method not only facilitates exposure and obviates the need of working

under the costal cage in a deep wound, but permits access to the portal vein to one side, instead of behind the common duct and hepatic artery. Briefly, the procedure is carried out as follows:

The patient is placed on the left side, in the true lateral position; a sand bag elevates the left loin. An incision is made over the ninth rib, commencing at the lateral border of the right erector spinae muscles and extending to the lateral margin of the right rectus muscle. The ninth rib is resected subperiosteally, from its angle to the costochondral juncture, which is severed. (The ninth interspace may be used.) After the chest is opened, the diaphragm together with the abdominal musculature and peritoneum are cut along the line of incision, thereby freely uniting the thoracic and abdominal cavities.

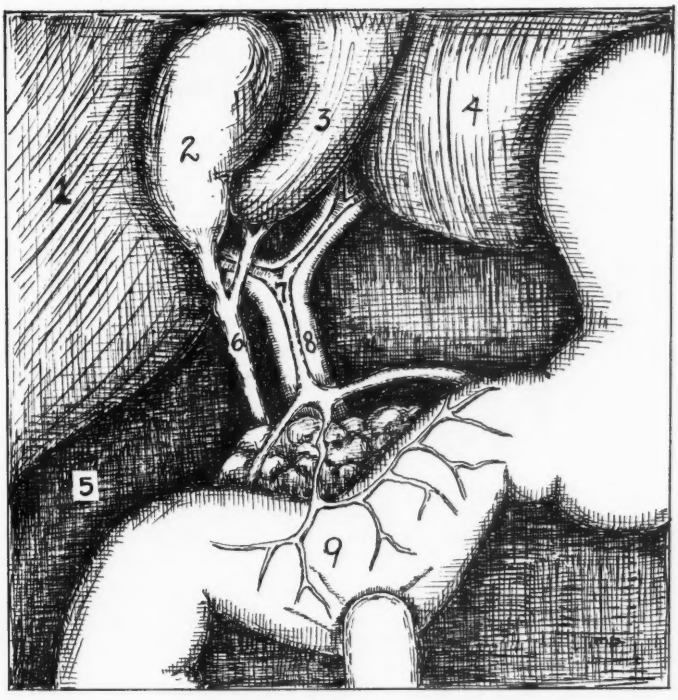

Fig. 3, *A*.

Fig. 3, *A* and *B*.—Schematic drawings comparing the relationship of the structures in the hepatoduodenal ligament as viewed from the prone (anatomic) position and the left lateral position.

A. Prone position (abdominal approach). The portal vein lies behind the common duct and hepatic artery: *1*, Right lobe of liver; *2*, gall bladder; *3*, quadrate lobe of liver; *4*, left lobe of liver; *5*, site of inferior vena cava; *6*, common bile duct; *7*, hepatic artery; *8*, portal vein; *9*, retracted duodenum exposing pancreas above.

The lung and intestines are packed away and the liver is dislocated into the right hemithorax, bringing the hilum of the liver and the inferior vena cava into direct view; handling of the intestines is minimal, thereby lessening the likelihood of postoperative ileus. The peritoneum over the inferior vena cava is incised, and the inferior vena cava is mobilized from the level of the liver to the entrance of the right renal vein. Attention is then directed toward mobilization of the portal vein.

It is wise to begin dissection of the portal vein laterally, so as to avoid injury to the common duct. Complete mobilization of the portal vein is required, from its origin at

the superior border of the pancreas to the points where its right and left branches enter the substance of the liver. Ligatures are placed around each branch and the main vessel is placed between the jaws of a Blalock clamp. At this time, pressure readings of the portal vein are taken, following which the Blalock clamp is tightened. The branches of the portal vein are then ligated. A transfixion ligature is placed at the site of bifurcation, and the portal vein severed transversely just below this ligature, ensuring the greatest possible length of portal vein. Traction on the portal vein now facilitates its complete mobilization.

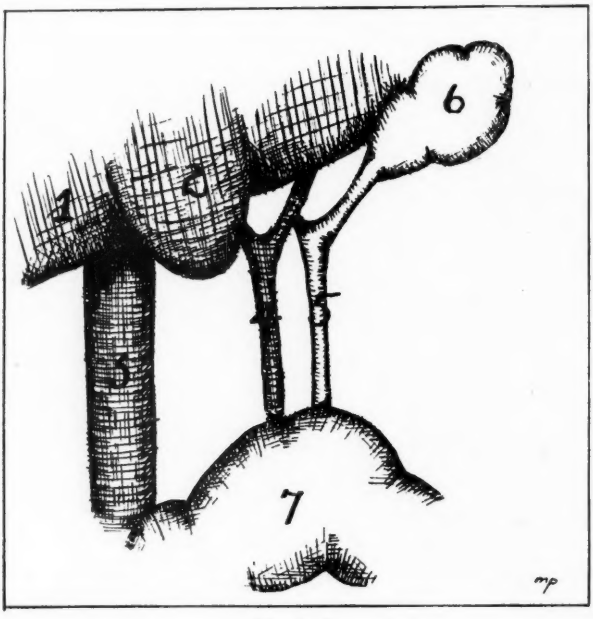

Fig. 3, *B*.

Fig. 3, *B*.—Left lateral position (thoracoabdominal approach). The portal vein now lies to one side of the common duct and hepatic artery; the common duct obscures the hepatic artery from view: *1*, Right lobe of liver; *2*, the caudate lobe of liver now comes into view; *3*, inferior vena cava; *4*, portal vein; *5*, common duct overlying the hepatic artery; *6*, gall bladder now lying to the right; *7*, duodenum.

The inferior vena cava has already been prepared for anastomosis. Since this vessel may not be clamped off above the level of the renal vessels for more than ten minutes without incurring anuria, it is necessary to permit vena caval blood flow during the anastomosis. This is made possible by merely constricting a portion of the anteromedial wall of the vessel by an unshod special clamp.* The pinched-off portion of the vessel is utilized for the anastomosis while blood flow beneath the horizontal blades of the clamp continues freely. A portion of the vein held in the clamp is then excised, the length of excision corresponding with the width of the severed portal vein. Excision of a portion of the vein is to be preferred over simple incision; the musculo-elastic fibers in the media of the inferior vena cava run longitudinally, and, if simply incised, would tend to reapproximate the wound. Since a transverse incision is not feasible, the removal of a small portion of the wall of the inferior vena cava insures against postoperative closure of the anastomosis. To facilitate this maneuver, a special long-handled scissors with a large, upward curve has been devised.*

*The Satinsky clamp and scissors are manufactured by George P. Pilling & Sons, Philadelphia, Pa.

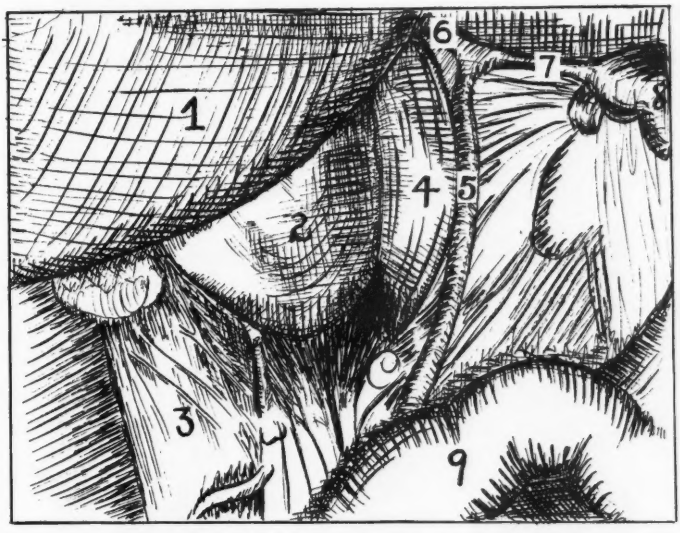

Fig. 4.—Following retraction of the liver and intestines, the operative area is exposed. 1, Retractor over inferior surface of right lobe of liver; 2, caudate lobe of liver; 3, inferior vena cava; 4, portal vein; 5, common bile duct; 6, hepatic duct; 7, cystic duct; 8, gall bladder; 9, duodenum.

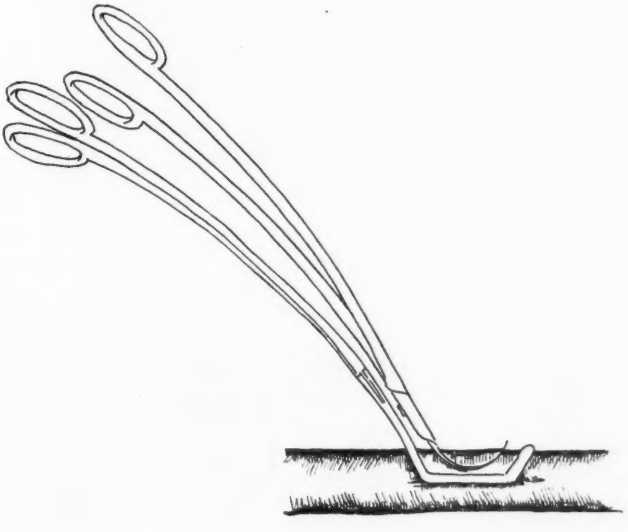

Fig. 5.—Satinsky clamp and scissors. The inferior vena cava is clamped in a manner that permits venous flow during the performance of the portacaval anastomosis. The special curve of the scissors facilitates excision of a portion of the inferior vena cava.

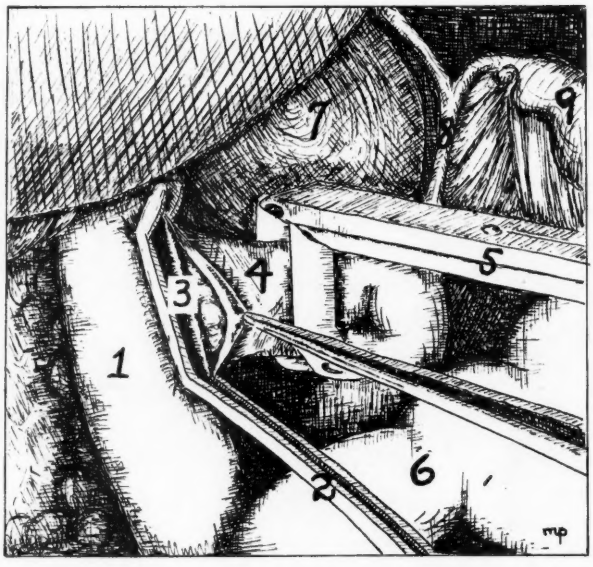

Fig. 6.—Clamps control portal vein and inferior vena cava as posterior layer of anastomosis is completed. *1*, Inferior vena cava; *2*, Satinsky clamp; *3*, posterior suture line; *4*, portal vein; *5*, Blalock clamp; *6*, duodenum; *7*, caudate lobe of liver; *8*, common bile duct; *9*, gall bladder.

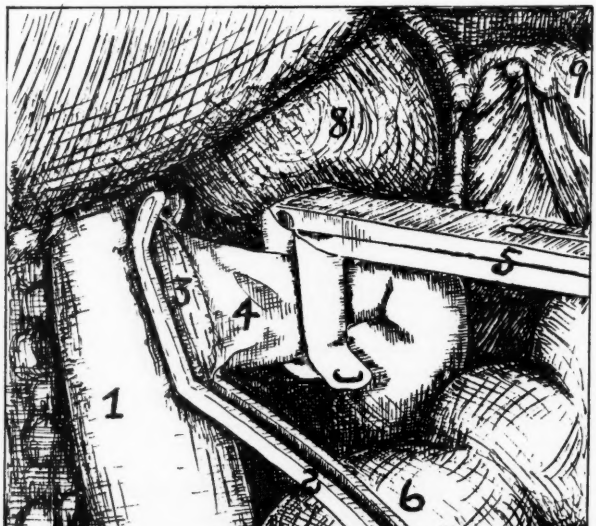

Fig. 7.—Anastomosis is completed with clamps still in place. The Blalock clamp will be released first. *1*, Inferior vena cava; *2*, Satinsky clamp; *3*, anterior suture line; *4* portal vein; *5*, Blalock clamp; *6*, duodenum; *7*, common bile duct; *8*, caudate lobe of liver; *9*, gall bladder.

Direct anastomosis of the proximal end of the divided portal vein to the side of the inferior vena cava is performed with 00000 braided silk on an atraumatic needle. A running everting mattress suture is used. The sutures are not intermittently interrupted as suggested by Blalock.[25] Instead, a knot is tied at the starting and end points of the posterior suture line to prevent purse stringing of the vessel. Both knots are tied on the outside. An interrupted stay suture unites the portal vein and inferior vena cava on either side of the suture line to relieve some of the tension at the angles of the anastomosis and to prevent the angles from pulling in. One of the free ends of the tension sutures is tied to a free end of the anastomotic sutures. Until the completion of the anastomosis, the intima of both vessels are frequently irrigated with a dilute heparin-saline solution.

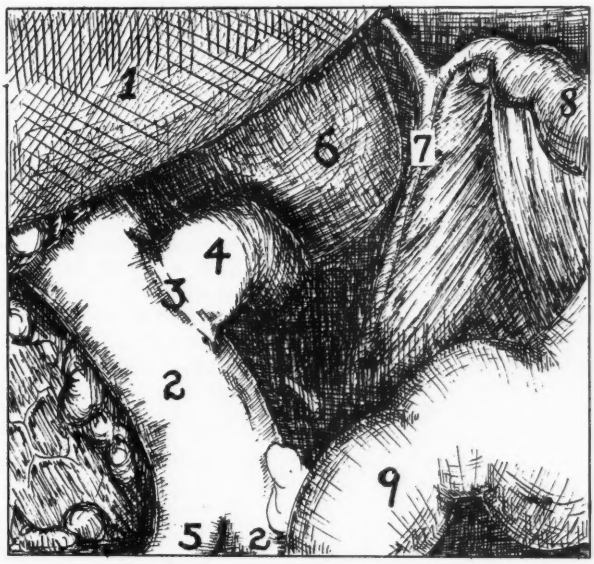

Fig. 8.—End-to-side, intima-to-intima anastomosis of the portal vein to the inferior vena cava; *1*, Retractor over right lobe of liver; *2*, inferior vena cava; *3*, suture line; *4*, portal vein; *5*, right renal vein; *6*, caudate lobe of liver; *7*, common duct; *8*, gall bladder; *9*, duodenum. Note position of suture line after release of clamps; the anastomosis occupies the anterior aspect of the inferior vena cava in a plane exceeding 90° relative to the right renal vein. There should be neither angulation or tension.

When the anastomosis is completed, the Blalock clamp is removed. This permits venous blood to fill up the interstices made by the sutures. Unless there is a large leak in the suture line requiring additional, interrupted sutures, the oozing will stop in a few moments. The clamp on the inferior vena cava then may be removed, and the efficiency of the shunt at once becomes visible. Penicillin is placed in the peritoneal and thoracic cavities (50,000 units into each), the diaphragm is sutured with imbricating, interrupted mattress sutures, and the abdomen and chest are closed in layers. At the Thoracic Surgical Unit of the Hahnemann Hospital, closure of the chest with intercostal drainage is the rule. Cotton suture technique is employed.

Because of the large size of the shunt, the fact that the portal tension is highest and the inferior vena caval pressure lowest at the site selected for anastomosis, and because a portion of the inferior vena cava is excised, there need be no fear of either postoperative closure or thrombus formation at the site of anastomosis. For this reason heparin is not employed postoperatively. Consequently, not only are the dangers of anticoagulant therapy avoided, but early ambulation is made possible.

An objection to the transthoracic route for portacaval shunt may be raised by those who argue the need for pressure readings of the portal system at the time of operation. Generally, a diagnosis of intra- or extrahepatic block can be made preoperatively. Indeed, at the present time, a good many patients have been found to have portal cirrhosis at a previous laparotomy. In any case, if pressure readings are desired, the abdominal portion of the wound can be made first, and the incision can be extended to the left instead of over the right costal cage, if a splenorenal shunt is specifically indicated. The use of a tilt table facilitates this maneuver.

CASE REPORTS

CASE 1.—E. G., male, aged 44 years, had enjoyed excellent health until he developed diabetes mellitus in 1940. Then, in January, 1946, the patient vomited blood and passed many tarry stools. A diagnosis of duodenal ulcer was made and purportedly confirmed by x-ray examination. An episode of hematemesis occurred eight months later and again in January, 1947. A splenomegaly was noted but apparently little significance was attached to this finding.

When the patient was first seen by one of us attention was focused upon the splenomegaly. Although the liver function tests were well within normal limits with the exception of the Bromsulphalein test, which showed 19 per cent retention, a diagnosis of cirrhosis of the liver was tentatively made.

In May, 1947, a laparotomy was performed. Cirrhosis of the liver was found, with marked splenomegaly; a biopsy of the liver was taken and a Talma-Morison procedure carried out. The presence of a duodenal ulcer was not demonstrated. The patient made an uneventful recovery and the pathology of the liver was reported as conclusive of extensive active portal cirrhosis.

Four weeks later another gastrointestinal hemorrhage took place. This was combated with blood transfusions.

From June, 1947, until January, 1948, the patient remained in poor health. He was maintained on periodic intramuscular injections of liver and 25 units of protamine zinc insulin. At this time the liver function tests were as follows: Total protein 6.4 with 4.2 albumin and 2.2 globulin; cephalin flocculation +1; thymol turbidity 4.3 units; Bromsulphalein 20 per cent retention in one hour; prothrombin time 55 per cent of normal; total cholesterol 250 mg. per cent; cholesterol esters 44 per cent. X-ray examination showed the presence of esophageal varicosities which until this time had not been demonstrated. Barium enema showed the splenic flexure to be sharply depressed by a huge spleen lying above.

A portacaval shunt for the relief of bleeding of esophageal varices which was secondary to an obvious portal hypertension seemed clearly indicated and on Jan. 28, 1948, the shunt was established according to the previously described method. A moderate degree of ascites was found at the time of operation. Convalescence was uneventful. Since operation the patient has enjoyed considerable comfort and there has been no evidence of gastrointestinal bleeding. The spleen is no longer palpable and there is no x-ray evidence of its enlargement. Esophagograms fail to demonstrate varices. As a whole, the liver function tests have remained unaltered. The patient has gained weight and with the exception of the diabetic state, does not consider himself ill or incapacitated.

Comment.—This case represents the ideal for transthoracic portacaval shunt: (1) The patient was in fairly good general condition; (2) liver function tests showed the liver not to be too severely damaged; (3) diagnosis had been unequivocably established; (4) maximum collateral circulation between the portal and systemic systems of veins had long been attained; its inadequacy was manifested by repeated bouts of gastrointestinal hemorrhage, and it appeared certain that only a more efficient collateral pathway, preferably in the form of

an Eck fistula, could avert a fatal outcome. At the same time, the clinical manifestations of a decompensating collateral circulation indicated that the liver was obtaining its blood supply almost entirely from a compensatorily dilated hepatic artery, and that it would, therefore, be safe to divide the portal vein; (5) an enormously enlarged spleen together with its highly vascular perisplenitis was left unmolested; (6) adhesions from the previous laparotomy and a previously elective attachment of the omentum to the anterior abdominal wall were completely avoided by the transthoracic thoracoabdominal approach.

In addition to the indications for portacaval shunt and the advantages of the recommended approach, this case emphasizes the desirability of preserving the spleen; congestive splenomegaly and its associated hypersplenism resolved shortly following the establishment of the portacaval shunt.

CASE 2.—A. A., male, aged 54 years, a heavy drinker, was admitted to the hospital in a state of shock one hour following an episode of severe hematemesis. Past history revealed hospitalization two months previously for hematemesis and progressive abdominal enlargement, a forty to fifty pound weight loss in the preceding two years, and variable ankle swelling during the preceding two months.

Significant Physical Findings on Admission.—Blood pressure 90/50, with a pulse rate of 124 per minute; grade 2 aortic systolic cardiac murmur; marked distention of the abdomen with elicitable fluid wave; nonpalpable liver and spleen; spider hemangiomas over the right chest and shoulder; bilateral 2+ ankle edema.

Significant Laboratory Studies.—Red blood cells, 2,000,000 per c.mm. with a hemoglobin of 7.8 grams per cent; white count essentially normal; blood urea nitrogen 28 mg. per cent; chlorides 479 mg. per cent; carbon dioxide combining power 40.9 volumes per cent; serum proteins 7.0 per cent with a 2.8 per cent albumin and 4.2 per cent globulin; Bromsulphalein excretion test 20 per cent retention; cephalin flocculation +2 in 48 hours; thymol turbidity test negative in 18 hours.

Course in Hospital.—Following admission the patient responded to liberal administration of citrated whole blood transfusions and plasma intravenously. He was placed on a high protein diet (200 grams daily), choline 1.0 Gm. three times daily, thiamine hydrochloride 100 mg., nicotinamide 100 mg. and ascorbic acid 500 mg. daily.

On the second, fourth, and sixth hospital days the patient vomited copious amounts of bright red blood; on each occasion he went into shock, but fortunately responded to liberal whole blood and saline infusions. X-ray examination of the esophagus with barium revealed esophageal varices and the patient was scheduled for portacaval shunt. But once again hematemesis supervened. In an attempt to arrest hemorrhage, esophageal tamponade was instituted, without success. Only repeated replacement of the blood loss kept the patient alive. By the eighth hospital day the patient was comatose, markedly icteric, and manifested auricular fibrillation. Blood urea nitrogen was 38 mg. per cent and the carbon dioxide combining power 31.5 per cent. Abdominal paracentesis at this time recovered 3,900 c.c. of amber fluid.

The patient remained in the state of apparent liver failure for seven days then slowly began to improve; laboratory findings returned to normal except for a red blood count of 2,800,000 per c.mm. with a hemoglobin of 7.6 grams per cent; prothrombin level (Quick) 53 per cent; total protein 5.5 with a 2.7 per cent serum albumin and a serum globulin of 2.8 per cent.

Transthoracic portacaval anastomosis was performed 24 days following admission. At operation a small hobnailed liver was demonstrated. During the course of the operation (a 4-hour procedure), the patient received 1,500 c.c. of citrated whole blood and left the operating room in good condition. On the first postoperative day the patient was

ambulated and hematologic studies were essentially unchanged from preoperative levels. On the second postoperative day, without warning, the patient experienced a sudden bout of severe hematemesis and despite replacement therapy died in two hours.

Pertinent autopsy findings revealed the portacaval stoma to be inviolate. However, there was marked compression of the portal vein which was, in part, found to be overlying the anterior aspect of the inferior vena cava proximal to the suture line. Other significant findings were a disproportionately enlarged caudate lobe of the liver and severe congestion of the major part of the gastrointestinal tract, with intermittent areas of small ruptured veins. The fatal gastrointestinal hemorrhage was secondary to the marked portal hypertension which followed upon compression of the portal vein. Undoubtedly, it was the preoperative, physiologic connections between the portal and systemic systems of veins which accounted for the forestalling of death until the second postoperative day.

Comment.—In retrospect it was easy to reconstruct the combination of anatomic factors and technical errors which resulted in compression of the portal vein: (1) In this patient the caudate lobe was inordinately enlarged, almost reaching the level of the renal veins. Unfortunately, the portal vein was permitted to overlie the caudate lobe in its new, anastomotic position; we realize now this was a mistake in judgment. But even this factor in all probability would not have had such disastrous effects were it not for the fact that (2) the suture line was established on the same plane as the right renal vein. This means that when the patient was rotated on his back, from the point of view of anatomic position the suture line occupied the true lateral, instead of the desirable anterior or medial aspect of the inferior vena cava. The portal vein, stretched over the inferior vena cava and subjected to an increase in intra-abdominal pressure in the prone position, was, in effect, sandwiched between two resistant forces. Again, if the caudate lobe were not enlarged, it is possible that the poor position of the suture line would not have had such a catastrophic result.

There are several lessons to be learned from this case:

1. A patient can be carried through an operation for portacaval shunt with prothrombin levels just above 50 per cent.

2. In the true lateral position, the inferior vena cava presents its lateral aspect, more or less in the plane of the right renal vein. Care must be taken to establish the anastomosis of the portal vein to the inferior vena cava in the plane 90° to 180° in relationship to the right renal vein; this precaution will guard against "twisting" of the portal vein over the inferior vena cava. The use of a tilt table may be helpful in securing the desirable position for anastomosis. In practice, however, the initially recommended position of the patient presents no real difficulty since the long, curved handle of the Satinsky clamp facilitates easy selection of a site for anastomosis and efficient control of the inferior vena cava.

3. If an enlarged caudate lobe is present, and in Laennec's cirrhosis this is an unusual finding, the transposed portal vein should be placed below this structure so that it can rest unencumbered on all sides. It may be necessary to deliver the portal vein below the pancreas and establish the anastomosis caudal to the level of the renal veins. As an alternative, the hindering portion of the caudate lobe of the liver may be resected.

4. The urgency of surgical intervention in cases of portal cirrhosis with severe hematemesis is emphasized. Undue delay invites repeated bouts of gastrointestinal hemorrhage. The optimal time for surgery is immediately following restoration of blood loss, other factors permitting.

CASE 3.—S. D., male, aged 47 years, was an acknowledged alcoholic who admitted to the consumption of one bottle of whiskey daily; anorexia, thirty-five pound weight loss, and progressive ascites and dependent edema during the two years prior to admission; edema of eyelids, severe dependent edema and ascites commencing eleven weeks before hospitalization, with an incident of coma during this time; repeated abdominal paracentesis.

Positive Physical Finding on Admission.—Emaciation, disorientation; liver edge palpable at the crest of the ileum; marked abdominal distention, and dependent edema.

Significant Laboratory Studies.—Serum proteins 6.1; albumin fraction 2.2 per cent; globulin fraction 3.9 per cent; prothrombin (Quick) 63.5 per cent; cephalin flocculation +3 in 18 hours; thymol turbidity +3; Bromsulphalein 15 per cent retention; blood urea nitrogen 10; chlorides 596.7.

Course in Hospital.—Following admission, patient was placed on a high protein diet (200 grams daily), crude liver 2 c.c. intramuscularly and Mercupurin 2 c.c. daily; Solulexin ampules 1 twice daily and Synkovite 10 mg. daily. Peritoneoscopy revealed a large, moderately nodular and granular liver. Biopsy specimens taken from the liver at this time failed to absolutely reveal the structural cellular pattern of cirrhosis. Despite maximum vigorous therapy, the patient showed no improvement. On the thirteenth day after admission, a transthoracic portacaval anastomosis was performed. The pressure in the portal vein prior to shunt was 400 mm. of H_2O; following anastomosis it was found to be 160 mm. of H_2O. The patient was administered 1,500 c.c. of citrated whole blood through a 3½-hour procedure, and left the operating room in good condition. The immediate postoperative course presented certain difficulties. The patient was uncooperative and refused to eat. Intravenous protein hydrolosates and whole blood transfusions were employed to maintain protein levels. Abdominal paracenteses were required at weekly intervals; between 2,700 and 5,000 c.c. of amber fluid were withdrawn. Four weeks postoperatively, in addition to dependent edema and ascites, the patient demonstrated a right hydrothorax; thoracentesis recovered 1,200 c.c. of fluid on one occasion and 1,900 c.c. on another. During this time total proteins ranged between 5.0 and 5.9 with albumin levels between 2.3 per cent and 2.2 per cent and globulin values between 2.7 per cent and 3.7 per cent. Approximately six weeks following the establishment of portacaval shunt the patient commenced to show improvement. He was discharged nine weeks following surgery without ascites, edema, or hydrothorax. At this time total protein was 5.3, albumin 3.2 per cent, globulin 2.1 per cent.

Comment.—This case brings out three points worthy of special consideration. (1) A blood serum albumin below 3 per cent is not necessarily a contraindication for portacaval shunt, as generally believed. This patient tolerated surgery well with an albumin of 2.2 per cent. (2) Patients with cerebral manifestations of liver failure in addition to portal hypertension should be operated upon as soon as their general condition permits. We have had instances in which "waiting for the optimal time" following recovery from cerebral involvement finally eventuated in a recurrence of symptomatology with death. We believe that *the optimal time is as soon as possible.* As in this case, patients may continue to go downhill despite adequate medical treatment. Actually, by ordinary standards, this patient was a very poor surgical risk. (3) Following portacaval shunt, ascites and edema may not disappear for several weeks. It is interesting

to note that there was very little change in the total protein and albumin-globulin ratios from the time of admission to discharge, certainly not enough to account for the hydrothorax and the subsequent gradual disappearance of the ascites and edema. This observation opens up for conjecture the possible existence of factors other than those already known governing tissue fluid distribution (W. Likoff).[26]

CASE 4.—R. W., aged 22 years, was hospitalized in a profound state of shock following an acute episode of hematemesis. Past history revealed that three years previously, following an attack of hepatitis, a diagnosis of Banti's disease was made. Exploratory laparotomy at that time demonstrated hepatic cirrhosis associated with splenomegaly. For one year before admission there had been intermittent edema of the legs and ankles, and progressive increase in abdominal girth. Four months prior to admission the patient experienced and ignored two bouts of gastrointestinal hemorrhage, expressed orally.

Laboratory Studies on Admission.—Red blood cells, 1,940,000 with a hemoglobin of 5.5 grams per cent; white blood count essentially normal; prothrombin time (Quick) 100 per cent; serum proteins 4.55 per cent with an albumin fraction of 2.91 per cent and a globulin fraction of 1.64 per cent; Bromsulphalein test showed 24 per cent retention of the dye; cephalin flocculation +3 in 48 hours; thymol turbidity was +1 in 24 hours; cholesterol 140.

Course in Hospital.—Pertinent physical findings on admission revealed a pale, apprehensive white man with subnormal temperature. Pulse rate was 134 and the blood pressure barely perceptible at the level of 50 mm. of mercury systolic. He made remarkable recovery with liberal whole blood transfusion. The patient was placed on a high protein diet with supplementary vitamin, liver, and lipotropic preparations. Studies of the esophagus by x-ray examination demonstrated considerable varicosities of the lower esophagus. With repeated blood transfusion a hematologic level of 4,000,000 red blood cells per c.mm. with a hemoglobin of 11.6 grams per cent was finally obtained.

Seventeen days after admission, a transthoracic portacaval anastomosis was performed. Pressure reading in the portal vein prior to anastomosis was 400 mm. of water. At the completion of the anastomosis pressure within the portal vein was 190 mm. of water. The patient was administered 2,000 c.c. of whole blood through the course of a 4-hour procedure and left the operating room in good condition. The postoperative course was uneventful. The patient was discharged on the twenty-first postoperative day, with the edema just beginning to disappear. Liver function studies at the time of discharge showed no appreciable changes.

Comment.—In this case, portal hypertension was clinically manifested by both ascites and gastrointestinal hemorrhage. A normal prothrombin time and an admission serum albumin above the critical level for edema demonstrated that the ascites and hemorrhage were not attributable to factors of depressed liver function; portacaval shunt was the only means of averting unexpected death from rupture of esophageal varices or expected wasting from loss of proteins in ascitic fluid.

SUMMARY

I. *The indications for portacaval shunt* may be summed up as follows:

1. Portal hypertension due to intrahepatic block in which gastrointestinal hemorrhage, ascites, or both threaten the life or comfort of the patient, immediately or in the predictable near future, and in which the hemorrhage or ascites are proved to be due to portal hypertension and not low prothrombin and serum albumin levels.

2. Over-all liver function studies which will offer the patient a fair chance of surviving surgery.

a. Patients with a prothrombin time of 50 per cent or more will tolerate surgery, from the point of view of a controllable hemorrhage diathesis.

b. Patients with serum albumin ranging in the low levels of 2.0 per cent will tolerate surgery, from the point of view of depressed liver function. It is naturally expected that every known means be utilized to improve liver function maximumly prior to operation.

3. Cases of intrahepatic portal block in which an enormously enlarged spleen, a previous splenectomy, and/or a previous abdominal laparotomy mitigate against a contemplated splenorenal shunt. These indications are included only for the sake of completeness, since it is our belief that splenorenal shunt is in general a less desirable procedure.

II. *Optimal time for surgery*

1. Patients with depressed liver function should be operated upon as soon as maximum improvement is reached following a vigorous medical regime; this should include the administration of human serum albumin in selected cases. If the medical regime is carried too long, the opportunity for surgery may be lost.

a. Exsanguinating hemorrhage may intervene.

b. Fatal cerebral involvement may occur.

c. Prothrombin values may begin to fall after obtaining a maximum level.

d. Serum protein values and albumin-globulin ratios may decline after reaching their peak.

2. Patients with fairly good liver function evidencing severe hematemesis should be operated upon as soon as possible following restoration of blood loss.

III. *Prognosis*

1. Patients surviving surgery can be assured relief from the effects of portal hypertension.*

a. Esophageal varicosities eventually disappear, both clinically and by barium study.

b. Congestive splenomegaly and hypersplenism resolve.

c. Ascites and edema attributable to portal hypertension clear up; several weeks may elapse before improvement is noted.

2. Liver function studies may show no change, improvement, or slight regression.

IV. *The precautions to be taken and the pitfalls encountered in the performance of transthoracic portacaval shunt* may be summed up as follows:

1. The site for suture line should lie on the anterior or anteriomedial aspect of the inferior vena cava, in a plane lying between 90° and 180° relative to the right renal vein; the employment of a Satinsky clamp facilitates proper selection and control of the anastomosis site; a tilt table may be useful.

*It is now two and one-half years since the first patient was operated upon. The spleen is nonpalpable and the patient has been completely free of symptoms; prior to surgery he had suffered a bout of hematemesis on the average of once every three months. The details of twelve additional cases will be reported upon in a subsequent paper.

2. Avoid angulation and/or tension of the suture line.

3. Care must be taken to place the transposed portal vein below an enlarged caudate lobe of liver. To accomplish this, resection of the caudal portion of the caudate lobe or anastomosis of the portal vein to the inferior vena cava below the level of the renal veins may be necessary.

4. Dissection of the portal vein is begun laterally so as to avoid injury to the common duct.

5. It is not necessary to shod the jaws of the Satinsky clamp. Shodding not only reduces available anastomosing space but interferes with accurate closure of the blades.

6. A portion of the inferior vena cava should be excised rather than simply incised.

7. Until completion of the anastomosis, a dilute heparin-saline solution should be employed locally to irrigate the intima of the inferior vena cava and portal vein.

8. Early ambulation is encouraged.

V. *The advantages of a transthoracic, thoracoabdominal portacaval shunt* are decisive and manifold and may be summed up as follows:

1. Exposure is excellent because of the free spread of the ribs, and the free communication of the chest and abdominal cavities.

2. The liver can be dislocated into the right hemithorax, since the restricting effect of the diaphragm is obviated. Access to the hilum of the liver is, therefore, direct and simple.

3. The structures in the lesser omentum are approached from the lateral aspect, advantageously presenting the portal vein to one side, instead of behind the common duct and hepatic artery.

4. Ligation of the right and left branches of the portal vein is possible. Such high ligation, flush with the liver, not only affords a longer segment of portal vein for anastomosis but offsets the chances of thrombus formation initiating in a blind venous pouch.

5. The inferior vena cava and the hilum of the liver are presented more superficially in the lateral than in the prone position.

6. Troublesome venous collateral circulation is more easily coped with by this direct exposure. Indeed, it is practically avoided, particularly the network within the hepatoduodenal ligament; these collaterals are often mistaken for "cavernomatous transformation of the portal vein," and frequently mislead the surgeon into abandoning attempts to establish a portacaval shunt.

7. By employing the thoracoabdominal route, portacaval shunts will be favored over splenorenal anastomosis. This is desirable for the following reasons:

   a. Portacaval shunt is more efficient.
   b. The function of the spleen is preserved.
   c. Failure to establish a portal-systemic anastomosis because of insurmountable perisplenitis is avoided.

d. Heparin is not required postoperatively as it is with splenorenal shunt. The large shunt and wide pressure gradients afforded by portacaval anastomosis, together with the precaution of excising a portion of the inferior vena cava insure against spontaneous closure of, or thrombosis at the suture line.

8. With the patient lying on the left side the suture line supports its maximum tension. The dependability of the suture line is, therefore, visualized immediately at the time of operation.

9. Because of the simpler technique of the thoracoabdominal approach, operative time is reduced by more than one-half, thereby avoiding the surgical shock associated with lengthy procedures and prolonged anesthesia.

10. The thoracoabdominal approach permits portacaval anastomosis to be carried out with minimum handling of intestine, thereby preventing or reducing considerably postoperative ileus.

11. Adhesions from a previous abdominal laparotomy or Talma-Morison operation are avoided. Moreover, there is less likelihood for the formation of anterior abdominal wall adhesions, with their attending complications.

12. The thoracoabdominal approach is less likely to give rise to incisional hernia, since the abdominal portion of the wound comprises a small part of the entire incision.

CONCLUSION

Thoracoabdominal approach for portacaval anastomosis appears to be the method of choice for the treatment of portal hypertension secondary to intrahepatic block. By means of this simplified approach, it is felt that portacaval shunt will achieve a more dynamic role in the therapy of portal hypertension.

REFERENCES

1. Drummond, D., and Morison, R.: Case of Ascites Due to Cirrhosis of the Liver Cured by Operation, Brit. M. J. **2**: 728, 1896.
2. Talma, S.: Chirurgische Offnung Neuer Seitenbahnen fur das Blut der Vena Porta, Berlin. Klin. Wchnschr. **35**: 833-836, 1898.
3. Crosby, Roy C., and Cooney, Edward A.: Surgical Treatment of Ascites, New England J. Med. **235**: 581-585, 1946.
4. Crafoord, Clarence, and Frenchner, Paul: New Treatment of Varicose Veins of the Esophagus, Acta otolaryng. **27**: 422, 1939.
5. Walters, W., Moersch, H. J., and McKemon, D. A.: Bleeding Esophageal Varices; Evaluation of Methods Directed Toward Their Control, Especially by Direct Injection of Sclerosing Solution, Arch. Surg. **41**: 91, 1940.
6. Rowntree, L. G., Zimmerman, E. F., Todd, M. D., and Ajac, John: Intraesophageal Venous Tamponage: Its Use in a Case of Varical Hemorrhage From the Esophagus, J. A. M. A. **135**: 630-631, 1947.
7. Som, M. L., and Garlock, J. H.: New Approach to the Treatment of Esophageal Varices, J. A. M. A. **135**: 628-629, 1947.
8. Barnofsky, I., and Wangensteen, O. H.: Obstruction of Splenic Vein Increases Weight of Stomach and Predisposes to Erosion or Ulcer, Proc. Soc. Exper. Biol. & Med. **59**: 234-238, 1945.
9. Phemister, Dallas B., and Humphreys, Eleanore M.: Esophageal Resection and Total Gastrectomy in the Treatment of Bleeding Varicose Veins in Banti's Syndrome, Ann. Surg. **126**: 397-410, 1947.
10. Von Eck, N. V.: The Ligature of the Portal Vein, Preliminary Communication, Voyenno-Med. J. **130**: Section 2, 1877.
11. Whipple, Allen O.: The Problem of Portal Hypertension in Relation to the Hepatosplenopathies, Ann. Surg. **122**: 449-475, 1945.

12. Whipple, Allen O.: The Rationale of Portacaval Anastomosis, Bull. New York Acad. Med. **22**: 251-253. 1946.
13. Blakemore, Arthur H., and Lord, Jere W., Jr.: The Technic of Using Vitallium Tubes in Establishing Portacaval Shunts for Portal Hypertension, Ann. Surg. **122**: 476-489, 1945.
14. Blakemore, Arthur H.: Portacaval Anastomosis, A Report of Fourteen Cases, Bull. New York Acad. Med. **22**: 254-263, 1946.
15. Blakemore, Arthur H.: Indications for Portacaval Anastomosis, Surg., Gynec. & Obst. **84**: 645-653, 1947.
16. Warthin, A. S.: The Relation of Thrombophlebitis of the Portal and Splenic Veins in Splenic Anemia and Banti's Disease, Internat. Clin. 4(20th Series) **189**: 226, 1910.
17. Welch, C. Stuart: A Technique for Portacaval Anastomosis (Eck Fistula), Surg., Gynec. & Obst. **85**: 492-494, 1947.
18. Kelsey, Mavis P., Robertson, Harold E., and Griffin, Herbert Z.: Portal Obstruction in the Syndrome of Splenic Anemia, Proc. Staff Meet., Mayo Clin. **23**: 195-200, 1948.
19. Linton, Robert R., Jones, Chester M., and Volwiler, Wade: Portal Hypertension; The Treatment by Splenectomy and Splenorenal Anastomosis With Preservation of the Kidney, S. Clin. North America **27**: 1162-1170. 1947.
20. McIndoe, A. H.: Vascular Lesions of Portal Cirrhosis, Arch. Path. **5**: 23-42, 1928.
21. Herrick, F. C.: An Experimental Study Into the Cause of the Increased Portal Pressure in Portal Cirrhosis, J. Exper. Med. **9**: 93-104, 1907.
22. Linton, Robert R.: Portacaval Shunts in the Treatment of Portal Hypertension, With Special Reference to Patients Previously Operated Upon, New England J. Med. **238**: 723-727, 1948.
23. Satinsky, Victor P.: Thoracoabdominal Approach for Portacaval Anastomosis; With a Case Report of Portacaval Shunt Employing This Method, Ann. Surg. **128**: 938-947, 1948.
24. Satinsky, Victor P.: Portacaval Shunt for Portal Hypertension: A New Approach, Rev. Gastroenterol. **15**: 333-334, 1948.
25. Blalock, Alfred: The Use of Shunt By-Pass Operation in the Treatment of Certain Circulatory Disorders, Including Portal Hypertension and Pulmonary Stenosis, Ann. Surg. **125**: 129-141, 1947.
26. Likoff, William B.: Personal communication.

DISTURBANCES IN ACID-BASE BALANCE DURING ETHER ANESTHESIA

With Special Reference to the Changes Occurring During Thoracic Surgery

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CONSIDERABLE interest has been directed lately to the disturbances in acid-base balance of the blood occurring as a result of ether anesthesia during intrathoracic procedures. Beecher and associates[3] demonstrated the development of a progressive respiratory acidosis in the course of lobectomies and pneumonectomies. This acidosis was frequently uncompensated in that a definite, and sometimes ominous, fall in arterial plasma pH was encountered. In a number of instances the respiratory acidosis was complicated by the accumulation of fixed acids and therefore some degree of metabolic acidosis was also present.

The mechanisms by which carbon dioxide is allowed to accumulate in the alveolar air during anesthesia are not entirely clear. It was in an effort to elucidate these mechanisms that the present study was undertaken. It is self-evident that only by a clear understanding of the nature of the abnormalities in gas exchange can appropriate measures be taken to prevent this undesirable aspect of anesthesia.

MATERIAL AND METHODS

All patients studied were premedicated with barbiturates followed by morphine or demerol and atropine. Induction of anesthesia was accomplished by means of sodium pentothal and nitrous oxide. The latter was discontinued as soon as unconsciousness was reached, after which a mixture of ether and oxygen was given during the remainder of the operation. Closed intratracheal anesthesia was used in all cases except in kidney operations in which the ether-oxygen mixture was administered by means of a mask, using a closed system.

Six different groups of patients were studied. Five of these, namely, pneumonectomies, lobectomies, esophagectomies, thoracoplasties and kidney operations, were performed with the patients in the lateral position. The sixth group consisted of general surgical procedures on patients lying in the supine position.

Arterial blood, both for control studies and for observations during anesthesia, was collected under anaerobic conditions. One anesthesia sample was withdrawn as late as possible before anesthesia was discontinued. If the operation lasted for more than one hour, two or more blood samples were usually

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collected. The blood was taken to the laboratory where its analysis was immediately begun. Oxygen content of whole blood and CO_2 content of anaerobically separated plasma were determined in the Van Slyke manometric apparatus. Since the blood collected during anesthesia contained ether, the usual gasometric procedures could not be used. Oxygen content of this blood was determined by the method of Shaw and Downing,[5] while plasma CO_2 was analyzed by the Austin technique.[1] Duplicate determinations of the former checked within 0.1 volumes per cent, of the latter within 0.15 m. mol per liter. The oxygen capacity was determined by analysis of the blood equilibrated with room air in a flask for 20 minutes. In the presence of ether the air in the flask was flushed out repeatedly with a moist stream of room air before equilibration was started. The analysis could then be carried out in the routine fashion.[4] The plasma pH was measured in duplicate by means of the Van Slyke and associates photometric application to the Hastings-Sendroy technique.[7] The Beckman spectrophotometer, Model B, was used. The hematocrit value was calculated from the oxygen capacity.[4] The plasma pCO_2 and blood buffer base were found with the aid of the nomogram of Singer and Hastings.[6]

RESULTS

The results are tabulated in Tables I through VI. They can be summarized as follows:

1. In the great majority of cases in all six groups, a total of 48, a fall in arterial pH and a rise in arterial CO_2 tension were observed in each of the

TABLE I. SIX PNEUMONECTOMIES (INTRATRACHEAL ETHER-OXYGEN)

PATIENT	DIAGNOSIS	TIME AFTER POSITION (HOURS)	TIME PLEURA OPEN	O_2 SATURATION (%)	PL. CO_2 (M.MOL./L)	PL. pH	PCO_2 (MM. HG)	B.B.B. (MEQ./L)
Po	Tbc	:0	:0	87.52	26.93	7.46	37	50
		:10	:0	100.0	23.62	7.08	70	37
		:40	:20	100.0	18.06	7.38	30	41
		1:10	:50	100.0	21.06	7.32	39	41
Bu	Ca	:0	:0	94.15	24.83	7.32	46	44
		:20	:0	94.60	25.78	7.25	55	44
		2:30	1:45	85.60	34.65	6.82	170 ±	36
		(operation—just over on back-breathing O_2)		100.0	24.85	7.18	62	41
Fu	Ca	:0	:0	89.44	25.60	7.43	37	47.5
		:45	:20	95.88	22.91	7.33	41.5	43.5
		1:10	:45	99.89	22.95	7.32	42	43
Mu	Ca	:0	:0	92.04	19.25	7.33	35	40
		1:23	1:00	96.60	21.22	7.13	60	37
		1:40	1:17	90.75	21.07	7.19	52	38
Co	Tbc	:0	:0	94.07	23.79	7.44	34	46
		2:00	1:20	90.58	24.15	7.33	44	44
		3:30	2:50	87.43	25.57	7.32	48	45
St	Tbc (post thoracoplasty)	:0	:0	91.96	29.09	7.47	39	52
		1:25	:55	91.19	30.91	7.27	65.5	49
		2:20	1:50	94.91	28.88	7.31	54	48
		3:35	3:05	89.41	28.70	7.33	52	48

TABLE II. SEVEN LOBECTOMIES (INTRATRACHEAL ETHER-OXYGEN)

PATIENT	DIAGNOSIS	TIME AFTER POSITION (HOURS)	TIME PLEURA OPEN	O_2 SATURATION (%)	PL. CO_2 (M.MOL./L)	PL. pH	PCO_2 (MM. HG)	B.B.B. (MEQ./L)
Wa	Ca	:0	:0	93.58	26.20	7.43	38	49
		:10	:0	100.0	29.62	7.25	63	46
		1:50	1:15	99.70	35.52	7.04	120	44
Se	Hamartoma	:0	:0	97.95	23.69	7.42	35	45
		:31	:06	97.81	27.58	7.29	55	46
		1:35	1:10	100.0	27.71	7.23	61.5	44
Lo	Tbc	:0	:0	93.49	27.85	7.44	39.5	49.8
		1:00	:35	100.0	28.77	7.25	62	45.5
Ma	Tbc	:0	:0	91.61	22.60	7.45	32	44
		1:30	1:05	87.69	25.79	7.23	58	43
		3:45	3:20	90.55	26.52	7.25	57	44
Le	Bronchiectasis	:0	:0	89.78	28.90	7.46	39.5	51
		1:25	1:05	93.73	33.75	7.23	75	49
Ch	Blastomycosis	:0	:0	94.99	24.08	7.47	32	47.5
		1:25	1:15	100.0	23.27	7.27	48	41
Ba	Cystic disease	:0	:0	87.72	28.61	7.40	44	50
	Bronchiectasis	1:30	1:12	100.0	39.28	6.96	170	43

samples withdrawn during anesthesia as compared with the control sample. In all but 7 of the 68 observations made during anesthesia, the arterial oxygen saturation was within normal limits.

2. All pneumonectomies (6 cases) and lobectomies (7 cases) showed this disturbance in acid-base balance. No significant differences were evident between the two groups. The greatest fall in pH encountered in each patient amounted to .11 and .39 units, except in one patient on whom a pneumonectomy was performed. This patient, a 50-year-old man (Bu) whose blood showed a fall in pH of 0.50 units, had had severe bleeding from the pulmonary artery just before the sample was taken. It is of interest that as long as 24 hours after the operation the pH was still 7.25. It had returned to normal on the third postoperative day.

The rise in arterial pCO_2 in the two groups under consideration amounted to 5 to 35.5 mm. Hg, except in Bu whose carbon dioxide tension mounted to 170 mm. Hg.

TABLE III. THREE ESOPHAGECTOMIES (INTRATRACHEAL ETHER-OXYGEN)

PATIENT	DIAGNOSIS	TIME AFTER POSITION (HOURS)	TIME PLEURA OPEN	O_2 SATURATION (%)	PL. CO_2 (M.MOL./L)	PL. pH	PCO_2 (MM. HG)	B.B.B. (MEQ./L)
Ow	Esophagitis	:0	:0	91.01	25.53	7.47	34	48
		1:08	:33	93.34	24.96	7.42	37	47
		4:10	3:35	88.02	25.94	7.42	38	49
Du	Ca	:08	:0	100.0	30.76	7.45	44	53
		1:50	1:25	96.94	30.95	7.42	47	52
				100.0	28.69	7.40	44	50
Hu	Ca	:0	:0	88.81	30.94	7.44	44	53
		2:55	2:20	100.0	27.58	7.31	52	47
		3:05	2:30	100.0	26.74	7.28	54	45

TABLE IV. THIRTEEN THORACOPLASTIES (INTRATRACHEAL ETHER-OXYGEN)

PATIENT	STAGE	TIME AFTER POSITION (HOURS)	O_2 SATURATION (%)	PL. CO_2 (M.MOL./L)	PL. pH	PCO_2 (MM. HG)	B.B.B. (MEQ./L)
Ge	1st (3 ribs)	:0	94.60	24.49	7.40	38	46
		1:00	100.0	22.45	7.37	37.5	---
Ma	2nd (3 ribs)	:0	94.77	28.54	7.42	42.5	50
		1:10	----	----	7.37	---	---
Ge	2nd (3 ribs)	:0	93.18	26.71	7.45	37	49
		:45	96.38	23.81	7.40	36.5	45
Po	Post pneumonectomy (4 ribs)	:0	93.28	27.99	7.51	34.3	51.5
		:28	100.0	26.26	7.40	41	47.5
Ha	2nd (3 ribs)	:0	91.43	26.90	7.35	46	47
		:43	100.0	24.66	7.28	47	44
Ma	3rd (4 ribs)	:0	87.01	28.14	7.41	43	49.5
		:33	100.0	27.17	7.35	47.5	46
Mo	1st (3 ribs)	:0	93.37	26.00	7.45	36	49
		1:18	100.0	22.73	7.44	32.3	46
		1:45	100.0	22.43	7.45	31.5	46
Du	1st (3 ribs)	:0	92.59	23.38	7.44	33.5	46
		1:10	100.0	23.08	7.30	44.5	43
Cl	1st (3 ribs)	:0	82.90	27.63	7.50	34.5	51
		1:20	83.77	28.11	7.32	52	47
Ta	1st (3 ribs)	:0	90.10	29.72	7.51	36	53
		1:40	100.0	24.92	7.47	32	48
Fi	1st (3 ribs)	:0	93.12	24.76	7.40	38.5	46
		1:30	94.12	22.98	7.35	40	44
As	1st (3 ribs)	:0	92.54	24.05	7.46	32.5	47
		1:25	100.0	26.91	7.40	41.5	47
Be	2nd (3 ribs)	:0	92.60	25.73	7.42	38	48
		1:00	100.0	22.26	7.36	38	44

TABLE V. EIGHT KIDNEY OPERATIONS (MASK ETHER-OXYGEN, CLOSED)

PATIENT	OPERATION	DIAGNOSIS	TIME AFTER POSITION (HOURS)	O_2 SATURATION (%)	PL. CO_2 (M.MOL./L)	PL. pH	PCO_2 (MM. HG)	B.B.B. (MEQ./L)
Mi	Left nephrectomy	Ca	:0	93.80	25.86	7.49	33	48
			:40	100.0	27.30	7.36	46	47
Ly	Left nephrectomy	Ca	:0	88.46	26.32	7.35	45	49
			:35	93.46	24.37	7.33	44	45
Su	Left nephrectomy	Pyelonephritis	:0	96.96	24.28	7.32	45	44
			1:05	100.0	23.46	7.23	53	40
Go	Right ureterostomy	Hydronephrosis	:0	91.50	31.33	7.43	47.5	51
			1:05	100.0	30.66	7.55	35	52
Ja	Removal left staghorn calculus	Kidney stones	:0	91.56	26.33	7.44	37.5	48
			1:00	100.0	27.99	7.27	57	45
St*	Removal tumor	Adrenal tumor	:0	89.36	28.11	7.49	36	50
			1:15	94.70	26.61	7.34	47	45
Br	Pyelolithotomy	Kidney stone	:0	91.77	24.98	7.42	37	47
			1:00	96.43	23.11	7.35	40	44
Ti	Right nephrectomy	Pyelonephritis	:0	91.34	23.47	7.42	35	45
			1:00	100.0	24.30	7.33	44	43

*Intubation in middle of operation.

TABLE VI. ELEVEN OPERATIONS ON BACK (INTRATRACHEAL ETHER-OXYGEN)

PATIENT	OPERATION	DIAGNOSIS	TIME AFTER INTUBATION (HOURS)	O_2 SATURATION (%)	PL. CO_2 (M.MOL./L)	PL. pH	PCO_2 (MM. HG)	B.B.B. (MEQ./L)
Bo	Craniotomy	Brain tumor	:0	93.36	26.01	7.49	34	50
			1:35	100.0	23.88	7.50	30	48
			3:00	100.0	23.57	7.45	33	47
Le	Subtotal gastrectomy	Ulcer	:0	94.66	24.35	7.43	35	46
			1:15	98.72	24.70	7.32	46	43.5
			2:40	100.0	20.85	7.33	38	41
El	Subtotal gastrectomy	Ulcer	:0	92.79	23.00	7.47	31	47
			2:00	97.69	24.77	7.33	44.5	45
			3:00	100.0	26.18	7.25	56.5	44
Gr	Subtotal thyroidectomy	Nontoxic goiter	:0	93.24	25.03	7.49	32	48
			1:30	100.0	23.19	7.34	41	44
Ro	Cholecystectomy	Gallbladder disease	:0	92.91	22.96	7.50	29	47
			1:25	----	22.27	7.38	36	--
Si	Subtotal thyroidectomy	Ca thyroid	:0	91.14	26.87	7.47	35.5	49
			1:00	100.0	25.80	7.39	41	46
Wi	Subtotal gastrectomy	Ulcer	:0	92.25	20.55	7.48	27	45
			2:20	100.0	32.32	7.05	108	42
Sh	Abdominoperineal resection	Ca rectum	:0	92.26	24.29	7.46	33	48
			1:20	96.34	27.23	7.31	51	47
			2:20	99.12	28.06	7.28	56	47
Fa	Right colostomy		:0	91.32	22.40	7.35	39	43
			:40	89.93	22.26	7.25	48	41
Po	Subtotal gastrectomy	Peptic ulcer	:0	96.43	29.02	7.42	43	52
			1:40	100.0	27.29	7.30	52.5	47
			2:15	----	28.16	7.22	64	45
Is	Abdominoperineal resection	Ca rectum	:0	95.12	25.08	7.44	36	46
			1:00	----	21.95	7.35	38	--
			2:05	100.0	26.66	7.22	61	42

Consistent evidence of the presence of a moderate degree of metabolic acidosis was found in the fact that the whole blood buffer base in all cases decreased to a maximum of 1 to 13 meq. per liter.

In those cases in which more than one blood sample was examined during anesthesia the changes in pH and pCO_2 were not always found to be progressive.

3. In three esophagectomies there was a maximal fall in pH of .05 to .21 units accompanied by a rise in pCO_2 of only 4 to 10 mm. Hg. These cases were under anesthesia for 2 to 4 hours, but better ventilation was maintained in them than in the lung resection cases.

4. The thoracoplasties (13 cases) and kidney operations (8 cases) can be considered together, as similar responses were encountered in the two groups. The arterial pH in these extrapleural procedures, carried out in the lateral position, fell to .01 to .17 units below the control observations. The arterial pCO_2 rose 1 to 19.5 mm. Hg in all but six instances in which a slight fall was observed. The blood buffer base fell 0 to 5 meq. per liter.

5. The blood findings were followed in 11 cases on whom general surgical procedures lasting from 40 minutes to 3 hours were performed. In 10 patients

the pH reached a level of .08 to .22 units below its control value; pCO_2 increased to 3 to 23 mm. Hg in excess of the preanesthesia level. One patient, a 53-year-old man with peptic ulcer, underwent a subtotal gastric resection in the course of which the pH fell to 7.05 and the pCO_2 mounted to 108 mm. Hg. This was the only observation made on this patient during anesthesia. The abnormalities were associated with a fall in systemic blood pressure.

The blood buffer base fell to 1 to 5 meq. per liter below its control value.

DISCUSSION AND CONCLUSIONS

The nature of the fixed acids which must have been released into the blood stream in each case of ether-oxygen anesthesia remains to be determined. The amounts of these acids were not greatly different in intrapleural procedures (average 4.7 meq. per liter) and in the remaining three groups of operations (average 3.0 meq. per liter). A consistent, though smaller, metabolic acidosis was also found by Beecher[2] in nonintrapleural procedures. On the other hand, his pneumonectomies and lobectomies showed nearly twice the degree of fixed acid accumulation found by us.

A degree of respiratory acidosis, as indicated by rising arterial CO_2 tension and falling pH, was present in most patients of each of the six groups. This was much more marked in the pneumonectomies and lobectomies. The most probable chain of events leading to the excessive increase in CO_2 tension of the alveolar air, and therefore of the arterial blood, is the following:

The respiratory center is primarily depressed by ether. This results in diminished alveolar ventilation with associated accumulation of CO_2. Far from having a stimulating effect upon the respiratory center, this increase in alveolar, and therefore arterial, pCO_2 further depresses a center already narcotized by ether. More impairment of ventilation follows; a vicious circle is thus set up leading to progressive accumulation of CO_2 in the alveolar air. That this chain of events does occur is confirmed by the observation that in none of the patients was an increase in ventilation observed during the course of anesthesia; on the contrary, the volume of ventilation seemed to be diminished in most instances.

It is therefore imperative that ventilation be *continuously* aided by extraneous means. This can consist of gentle and rhythmic manual kneading of the anesthesia bag or by any mechanical device which renders the same service. Failure to do this will be more harmful in pulmonary procedures than in extrapleural ones. The deflated state of the exposed lung, with maintenance of some blood flow through it, leads per se to accumulation of CO_2 in the arterial blood. However, it should be clear that keeping the lungs inflated is in itself not enough to permit adequate gas exchange.

The fact that in a number of patients some improvement in acid-base balance was observed as the operation progressed must be due to more effective kneading of the anesthesia bag, thereby leading to a temporary fall in alveolar pCO_2.

As the gas mixture administered contained more than 90 per cent oxygen, deficiency in arterial oxygen saturation should not be anticipated, even though

alveolar ventilation is inadequate. It must be emphasized that no cyanosis is therefore expected under those conditions.

Brief mention should be made of other possible mechanisms leading to CO_2 accumulation. The intratracheal catheter itself does not increase the dead space. In the absence of extraneous assistance to ventilation the "pendelluft" (air moving from the operated lung into the healthy one and vice versa) might result in an increase of physiologic dead space, but this effect is probably small. The lateral position in which the patient is placed in all intrapleural operations studied in this series has been suspected by Beecher of leading to respiratory acidosis.[3] Comparison of our results in thoracoplasties and kidney operations with those in operative procedures carried out with the patient in the supine position, fails to substantiate this possibility.

REFERENCES

1. Austin, J. H.: A Note on the Estimation of Carbon Dioxide in Serum in the Presence of Ether by the Van Slyke Method, J. Biol. Chem. **61**: 345, 1924.
2. Beecher, H. K., Francis, L., and Anfinsen, C. B.: Acid-base Balance During Ether Anesthesia in Man, J. Pharmacol. & Exper. Therap. **98**: 38, 1950.
3. Beecher, H. K., and Murphy, A. J.: Acidosis During Thoracic Surgery. J. THORACIC SURG. **19**: 50, 1950.
4. Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry; Methods, Vol. II, Baltimore, 1932, Williams and Wilkins Company.
5. Shaw, J. L., and Downing, V.: The Determination of Oxygen in Blood in Presence of Ether by a Modification of the Van Slyke-Neill Technique, J. Biol. Chem. **109**: 405, 1935.
6. Singer, R. B., and Hastings, A. B.: An Improved Clinical Method for the Estimation of Disturbances of the Acid-base Balance of Human Blood, Medicine **27**: 223, 1948.
7. Van Slyke, D. D., Weisinger, J. R., and Van Slyke, K. K.: Photometric Measurement of Plasma pH, J. Biol. Chem. **179**: 743, 1949.

CONTINUOUS SPINAL ANESTHESIA FOR THORACOPLASTY

Second Report

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In 1947, one of us (G. W.),[1] reported a series of 300 thoracoplastic operations performed under continuous spinal anesthesia. These cases were operated upon from February, 1941, to December, 1944. In the ensuing four-year period, the same anesthesia was used for all thoracoplasty operations. In view of the fact that it has continued to be satisfactory for both patient and surgeon, we believe it warrants another report.

Induction of continuous spinal anesthesia was attempted in 574 thoracoplasty operations during the four-year period ending December, 1948. These operations were done on 219 patients suffering from pulmonary tuberculosis. There were 115, or 52 per cent, male patients and 104, or 48 per cent, female patients, ranging in age from 17 to 60 years, with more than 60 per cent being under 40 years of age.

As an aid to comparative study, the cases have been divided into three groups: (1) unilateral pulmonary tuberculosis, 195 cases; (2) bilateral pulmonary tuberculosis, 237 cases; and, (3) bilateral pulmonary tuberculosis with contralateral pneumothorax, 142 cases. In addition to a stage of thoracoplasty, 87 patients had a Semb extrafascial apicolysis performed.

TECHNIQUE

The technique, as described in the first report, has remained essentially the same and only the salient steps will be repeated.

The patient is placed on the table in the position to be maintained during operation and should be moved as little as possible. Ephedrine sulfate, ⅜ grain, is given hypodermically and a skin wheal is raised in the lumbar region over the widest palpable interspace. A No. 19 gauge malleable spinal needle is used. If a free flow of spinal fluid is not obtaind, or if the fluid is bloody, a second puncture is made at another interspace. If either of these conditions still prevails, the operation is postponed. *A free flow of clear spinal fluid is absolutely essential.* Twenty cubic centimeters of spinal fluid is slowly withdrawn and is used to dissolve 500 mg. of procaine—2½ per cent solution. The needle is bent to the desired position and connected with the flexible rubber tubing. The tubing is then fixed to the patient's skin with adhesive tape. The operating table is lowered 10 to 15° at the head.

An initial dose of 100 mg. is administered and subsequent 50 mg. doses of procaine are given at two-minute intervals until the desired level of anesthesia is reached. It is rarely necessary in a first-stage thoracoplasty to give more procaine after the level of anesthesia has reached the mid-portion of the scapula. While the patient is prepared and draped, the anesthesia level will rise to the angle of the jaw. After closure of the muscle layers, the stopcock on the tubing is opened and spinal fluid withdrawn in the amount given as an anesthetic solution. This hastens the return of motor and sensory functions so that by the time the skin incision is closed and dressings are applied, our patient is able to move the lower extremities and has return of tone to the abdominal muscles. The spinal needle is not

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withdrawn until the level of sensation is at the lower angle of the scapula and the abdominal muscle tone or the ability to move the lower extremities is present.

The thoracoplasty patient, who has been anesthetized with continuous spinal anesthesia and has this degree of return of function, does not go into shock, remains cooperative, and is able to cough up secretions without difficulty. The postoperative nursing care, which is very difficult to obtain in sanatoriums, is greatly simplified.

Oxygen is given by mask to all patients as soon as the operation is begun and whole blood is transfused during this procedure as in any other major operation.

DOSAGE

The initial dose is usually 100 mg. of procaine and is followed with supplemental doses of 50 mg. at two- to three-minute intervals until the desired level is reached (the angle of the jaw for a first-stage thoracoplasty). Occasionally, subsequent doses must be given during the operation to maintain the desired level of anesthesia. The total dosage in this series has varied from 150 mg. to 2,200 mg. of procaine. However, 65 per cent of the stages have been done with a total dosage of procaine ranging from 150 mg. to 500 mg.

A 2½ per cent solution of procaine has proved to be the most satisfactory from the standpoint of a reasonably prompt induction as well as the matter of safety. The safety factors mean the observation of at least a two-minute interval before administering an additional amount of drug. In this series, 5 cases, or 0.8 per cent, had respiratory distress which could be attributed, at least in part, to the anesthetic. All responded promptly to withdrawal of the drug, positive pressure oxygen, and artificial respiration to aid the removal of the carbon dioxide.

EFFECT ON PULSE, BLOOD PRESSURE, AND RESPIRATION

An analysis of the statistics sumarized in Table I reveals no appreciable difference in the effect of spinal procaine on the pulse rate, blood pressure, or respiration of the three groups of patients studied. A diminution in pulse rate averaging 14 per minute was shown by 13.8 per cent of cases in Group 1 at the conclusion of operation. In Group 2, 16 per cent showed a fall averaging 17 per minute and 12 per cent of cases in Group 3 showed a fall averaging 19 per minute. A rise in pulse rate averaging 19 per minute was shown by 14.1 per cent in Group 1. In Group 2, 17.6 per cent of cases showed a rise averaging 19 per minute and 9½ per cent of the cases in Group 3 showed a

TABLE I

GROUP	PULSE				BLOOD PRESSURE				RESPIRATIONS				OPERATIONS DISCONTINUED
	FALL	RISE	NO CHANGE	NO RECORD	FALL	RISE	NO CHANGE	NO RECORD	FALL	RISE	NO CHANGE	NO RECORD	
1. Unilateral 190 cases (33%)	79	81	25	5	114	11	8	57	18	110	28	34	5
Percentage	13.8	14.1	4.3	0.8	19.8	1.9	1.4	9.9	3.1	19.2	4.8	5.9	0.8
2. Bilateral 224 cases (39%)	92	101	29	2	152	14	7	51	24	134	38	28	13
Percentage	16	17.6	5	0.3	26.5	2.4	1.2	8.8	4.2	23.4	6.6	4.8	2.2
3. Bilateral with pneumothorax 136 cases (24%)	70	55	9	2	87	11	5	33	8	96	26	6	6
Percentage	12	9.5	1.6	0.3	15.1	1.9	0.8	5.8	1.4	16.7	4.5	1	1

rise averaging 18 per minute. No change in pulse rate was recorded in 4.3 per cent of cases in Group 1, 5 per cent of Group 2, and 1.6 per cent of Group 3. There was incomplete data in 9 cases.

Complete records pertaining to blood pressure were available in 133 cases of Group 1, 173 cases of Group 2, and 103 cases of Group 3. A decrease in blood pressure was shown by 19.8 per cent of these cases in Group 1 at the conclusion of operation. In Group 2, 26½ per cent showed a fall and in Group 3, 15.1 per cent showed a fall. A rise in blood pressure was noted in 1.9 per cent of these cases in Group 1. In Group 2, 2.4 per cent and in Group 3, 1.9 per cent showed a rise in blood pressure at the conclusion of operation. No change in blood pressure was recorded in 1.4 per cent of cases in Group 1, 1.2 per cent of Group 2, and 0.8 per cent of Group 3.

Thirteen patients, or 2.2 per cent, went into a shocklike state during continous spinal anesthesia. The degree of shock was determined entirely by a markedly diminished blood pressure and an elevated pulse. However, the skin remained warm and dry in every instance. All responded promptly to withdrawal of the drug and transfusion of whole blood.

Complete data pertaining to respiration were recorded in 156 cases of Group 1, 196 cases of Group 2, and 130 cases of Group 3. A fall in respiration averaging 5 per minute was noted in 3.1 per cent of these cases in Group 1. In Group 2, 4.2 per cent showed a fall averaging 4 per minute and 1.4 per cent of cases of Group 3 showed a fall averaging 4 per minute. A rise in respiratory rate averaging 6 per minute was noted in 19.2 per cent of cases in Group 1. In Group 2, 23.4 per cent showed a rise averaging 7 per minute and 16.7 per cent of cases in Group 3 showed a rise averaging 8 per minute. No change in respiratory rate was noted in 4.8 per cent of cases in Group 1, 6.6 per cent of Group 2, and 4.5 per cent of Group 3.

COMPLICATIONS

There was no appreciable difference in the incidence of complications in the three groups of patients studied. During the operative procedure, 18 per cent of the patients complained of nausea and 2 per cent of vomiting or retching. All but 3 patients, or 0.5 per cent, were immediately relieved by withdrawal of spinal fluid usually 2 to 4 c.c. in amount.

In the previous report,[1] 4⅓ per cent of our patients complained of nausea and 13 per cent had vomiting. We believe the reversal of figures can probably be attributed to the fact that we now recognize the symptoms earlier and also to the use of a more dilute solution.

Postoperative headache occurred in 32 cases or 5.7 per cent. In none of these was the headache severe and in only one was it persistent, lasting for one week. Postanesthetic urinary retention, though of short duration, occurred in 2 patients, requiring catheterization.

Two patients developed a myeloradiculitis postoperatively. The first patient had a lower motor neurone paralysis on the right side with a level at the ninth thoracic dermatomere. She showed gradual improvement during the

postoperative course but on a follow-up visit one year later, there was still evidence of a partial paralysis of the right leg. The second patient showed evidence of a complete transverse lesion of the spinal cord at the level of the twelfth thoracic dermatomere. This occurred approximately twenty-four hours postoperatively. There was some improvement during the immediate postoperative period as evidenced by a return of sensation in the first, second, and third lumbar dermatomeres. One month following operation, this patient died of a pulmonary embolus.

In the preceding case, the paralysis occurred following the third stage of thoracoplasty under continuous spinal anesthesia and the operative procedure in each instance, as well as the anesthetic, was uneventful. The patient was able to move the lower extremities at the conclusion of each stage. The time interval between stages was three weeks. At the first stage, 750 mg. of procaine were used, at the second, 450 mg. and at the third, 900 mg. of procaine had been administered. This is an unusually large dose for a third-stage operation and there is no reason given in the record explaining this fact nor is there any time chart showing the intervals. It was noted that no bloody fluid was obtained. The free flow, however, was intermittent and lack of a constant free flow is an absolute contraindication to the administration of any spinal anesthetic. (We were subsequently informed that the patient had been under treatment for some years for syphilis, but this was not noted on the hospital record. Whether or not the syphilitic infection played a part in causing the myeloradiculitis is debatable.)

In 24 cases it was necessary to discontinue the operation because of difficulty with the induction of the anesthetic or unsatisfactory condition of the patient. These cases are listed as failures of this particular type of anesthesia, although, in some instances, other factors prevailed. These failures are briefly summarized in the following paragraphs.

CASE 1.—Unilateral pulmonary tuberculosis. Free flow of spinal fluid could not be obtained. Satisfactory level was not obtained after 700 mg. of procaine had been given. (No drug should have been given. Error in technique and judgment.)

CASE 2.—Unilateral pulmonary tuberculosis. Operation canceled because of bloody spinal tap. (Error in technique.)

CASE 3.—Unilateral pulmonary tuberculosis. Four taps were made but free flow of spinal fluid could not be obtained. (Error in technique.)

CASE 4.—Unilateral pulmonary tuberculosis. Spinal fluid bloody; 500 mg. of procaine given but no anesthesia obtained. (Error in technique and judgment.)

CASE 5.—Unilateral pulmonary tuberculosis. Three taps made but bloody fluid obtained each time. (Error in technique.)

CASE 6.—Bilateral pulmonary tuberculosis. Satisfactory level was not obtained after 480 mg. of procaine had been given. (No record as to free flow. Error in technique and judgment.)

CASE 7.—Bilateral pulmonary tuberculosis. Patient's blood pressure fell to 55/45 after 175 mg. of procaine had been given. (Patient in very poor general condition. Error in judgment.)

CASE 8.—Bilateral pulmonary tuberculosis. Patient received 900 mg. of procaine in first thirty minutes in order to obtain satisfactory level. Respirations became shallow and difficult. Pulse rose to 140 and blood pressure dropped to 60/50. (Excessive dosage in a short interval. Error in judgment.)

CASE 9.—Bilateral pulmonary tuberculosis. Satisfactory level could not be obtained. Later it was discovered that the patient received just a fraction of the dose it was thought he received, as new tubing contained 6 c.c. instead of the usual 2 c.c. (Error in technique.)

CASE 10.—Bilateral pulmonary tuberculosis. Patient had pain in left groin, leg, and foot following tap. No free flow of spinal fluid obtained. Satisfactory level not obtained after 300 mg. of procaine had been given. (This patient fortunately did not develop a myeloradiculitis. Error in technique and judgment.)

CASE 11.—Bilateral pulmonary tuberculosis. Marked paradoxical breathing developed after segment of rib removed and operation was discontinued. (No apparent error in technique or judgment.)

CASE 12.—Bilateral pulmonary tuberculosis. Pulse rose to 180, fifteen minutes after 150 mg. of procaine were given. Tachycardia persisted despite all effort to overcome it and surgery, therefore, was canceled. (Patient's pulse rate was 140 at the time of the induction of the anesthetic. Grave error in judgment.)

CASE 13.—Same patient as preceding one. Pulse rose to 192 after anesthetic was begun. Supplemental sodium pentothal analgesia was induced but tachycardia persisted and operation was canceled. (Error in judgment repeated.)

CASE 14.—Bilateral pulmonary tuberculosis. After 150 mg. of procaine had been given, pulse rose to 160 and blood pressure dropped to 80/65. Operation canceled. Patient promptly responded to withdrawal of drug, oxygen, and transfusion of whole blood. (Very poor risk. Error in judgment.)

CASE 15.—Bilateral pulmonary tuberculosis. Operation canceled because of bloody spinal taps. (Error in technique.)

CASE 16.—Bilateral pulmonary tuberculosis. After 250 mg. of procaine had been given and before surgery started pulse rose to 150 and respiratory rate to 36. Patient responded to transfusion of blood and withdrawal of drug. Operation canceled. (No note as to free flow. Error in technique and judgment.)

CASE 17.—Same patient as preceding one. Induction of anesthesia with a pulse rate of 120. After 200 mg. of procaine had been given and the skin and muscles had been incised, the pulse rate rose to 170 and blood pressure was recorded as 90/60. Operation immediately discontinued. (Error in judgment repeated.)

CASE 18.—Bilateral pulmonary tuberculosis. After 100 mg. of procaine had been given and before surgery was begun, the pulse rose to 165 and blood pressure dropped to 80/65. Operation immediately canceled. (No error in technique or judgment.)

CASE 19.—Bilateral pulmonary tuberculosis with contralateral pneumothorax. Patient had respiratory arrest following mobilization of scapula. Prompt recovery with oxygen, artificial respiration, and withdrawal of spinal fluid. (A total of 500 mg. of procaine had been given. However, without checking the anesthetic level again, supplementary doses were given after the desired level had been reached. Error in judgment.)

CASE 20.—Bilateral pulmonary tuberculosis with contralateral pneumothorax. Blood pressure dropped to 30 systolic after 400 mg. of procaine had been given. Prompt recovery with transfusion of whole blood, withdrawal of drug, and administration of oxygen under pressure. (Patient had an overdosage in a short time interval. Error in judgment.)

CASE 21.—Bilateral pulmonary tuberculosis with contralateral pneumothorax. After skin and muscle layers were incised, the pulse rate rose to 120 and blood pressure dropped to 80/60. Operation discontinued. (Error in judgment.)

CASE 22.—Bilateral pulmonary tuberculosis with contralateral pneumothorax. Patient had no anesthesia after 650 mg. of procaine had been given, although a good flow was present. Patient became very nervous and apprehensive and operation was canceled. (Very uncooperative. This case should never have been tried. Error in judgment.)

CASE 23.—Bilateral pulmonary tuberculosis with contralateral pneumothorax. It was impossible to get the anesthetic level above the eighth interspace. Operation completed under local anesthesia. (No note of free flow. Patient had a total dosage of 725 mg. of procaine. Error in technique and judgment.)

CASE 24.—Bilateral pulmonary tuberculosis with contralateral pneumothorax. After 150 mg. of procaine had been given and before surgery started, pulse rate rose to 160. Operation deferred. (No error.)

In summarizing the 24 failures, we note that 21, or 87½ per cent, are due to errors in technique or judgment or both and in only 3 cases could no error be detected. A large percentage of these failures occurred in the hands of the Junior Residents; hence, we wish to point out that with improvement in skill and judgment, failures will be minimal.

SUPPLEMENTARY ANESTHESIA

In this series it was necessary to use one or more supplementary anesthetic agents in 28 cases. These agents are listed in Table II.

TABLE II

	CASES
½% or 1% procaine by local infiltration	21
Nitrous oxide	2
Local infiltration and sodium pentothal	2
Cyclopropane	1
Cyclopropane and paravertebral block	1
Nitrous oxide and ether	1

The percentage of operations thus requiring supplemental anesthesia was 4.8: the effectiveness of continuous spinal anesthesia in this series being 95.2 per cent. When the 24 cases which were discontinued are added to this group, the total effectiveness of the continuous spinal anesthetic method is 91.2 per cent.

MORTALITY

There were 2 deaths in the operating room in this series which are briefly described in the following paragraphs.

The first patient was a 36-year-old woman with bilateral, far-advanced pulmonary tuberculosis with pneumothorax on the left side. Thoracoplasty was attempted on the right side. The anesthetic was administered without incident and the patient received 390 mg. of procaine during the one and one-half-hour operative procedure. The only ill effect noted was rather severe nausea, which was relieved by withdrawal of spinal fluid. During the operation, a traumatic pneumothorax was induced by a tear in the pleura. The intrapulmonic pressure was increased but the lung could not be completely re-expanded. The defect was then closed and air was aspirated, thus establishing a good negative pressure but the disturbed pulmonary function in

this seriously ill patient could not be restored. The respirations became increasingly more shallow and finally ceased. No drug was withdrawn.

The second patient was a 28-year-old man with bilateral, advanced pulmonary tuberculosis with pneumothorax on the right and cavitation on the left side. The anesthetic was administered with difficulty. The patient received 550 mg. of procaine in thirty-four minutes. Severe paradoxical motion with poor pulmonary ventilation developed after portions of the second and third ribs were resected. The operation was immediately discontinued. Withdrawal of drug was begun, tracheal intubation was also done, and artificial respiration administered. The airway was clear and in spite of artificial respiration, cyanosis increased, respirations became more shallow and then ceased.

Both of these patients were very poor risks with reduced pulmonary function as a result of the extent of the disease on the operative side as well as the reduced function in the contralateral lung due to artificial pneumothorax. Both deaths should be classified as due to errors in technique and judgment. Both of these anesthetics were administered by Junior Residents.

ANALYSIS OF HEADACHES

The cause and management of postspinal headache have been a subject of much thought and investigation for many years.[2, 3, 4] The dosage of the anesthetic agent is thought to play no part in its production.[5] Such headache has been ascribed by a minority to increased intracranial pressure and by a majority to decreased intracranial pressure.[6] Some claim that faulty technique and the size of the needle are contributory factors.

As previously stated, 32 patients, or 5.7 per cent, developed postspinal headaches in this series. This is not a large number of patients nor should it mitigate in any way against the use of this type of anesthesia. Watson[7] quoted Waters as analyzing 40,000 cases of cyclopropane anesthesia and finding the incidence of postanesthetic headache to be 10 per cent.

A No. 19 gauge malleable spinal needle is used and is kept in situ from one to three hours. Our patients are immediately returned to bed following surgery and instructed to lie flat for six to eight hours.

In 29, or 90 per cent, of the patients with headache, the technique was excellent in that a single tap was made and a free flow of clear spinal fluid was obtained. In 3 cases, multiple punctures were necessary before the anesthetic could be started. Thus, it would seem that in this series faulty technique did not play much of a role in the production of headache. Spinal fluid pressures were recorded in 25 of the 32 cases of headache. In 5 cases, or 20 per cent, the spinal fluid pressures recorded at the end of the operation were increased above normal. In 8 cases, or 32 per cent, the final spinal fluid pressures were decreased below normal and in 12 cases, or 48 per cent, the final spinal fluid pressures were within the normal range. Faulty technique, the amount of drug used, and the change in spinal fluid pressure, therefore, seem to bear no significant relation to the incidence of postspinal anesthetic

headache. The one common factor was the state of apprehension and anxiety during the preoperative period.

ADVANTAGES

The advantages of continuous spinal anesthesia for thoracoplasty are those of an anesthetic method that affords the patient maximum safety and comfort during the operation and minimal discomfort postoperatively. These patients are quiet, relaxed, and have even, steady respiratory excursions during the operative procedure. Anoxia, cyanosis, and paradoxical motion of the lung is at a minimum since the respiratory reserve of these chronically ill patients is not compromised by the added burden of an inhalation anesthesia. On return from the operating room, the patient is alert, can move about in bed, and is able to cough up secretions. Return to a full diet occurs usually the next day and is much more rapid than following a general anesthetic. There have been no postoperative pulmonary complications related to the anesthetic in any way in this series. The complete absence of the distressing postanesthetic prostration seen so frequently with inhalation anesthesia aids immeasurably in the prompt uneventful operative recovery of these patients.

CONCLUSIONS

1. Continuous spinal is the anesthetic method of choice for extrapleural thoracic procedures from the standpoint of patient safety and better surgical performance.

2. The technique is easily mastered and the effort expended is rewarded by a prompt operative recovery and a smoother postoperative course.

3. The complications occurring with this method are fewer than following inhalation anesthesia. They can be overcome in a great majority of cases by intelligent and skillful management.

4. Postoperative nursing requirements are reduced to a minimum.

REFERENCES

1. Willauer, G. J., Chodoff, R. J., and Garcia-Oller, J. L.: Continuous Spinal Anesthesia for Thoracoplasty, J. THORACIC SURG. **16**: 438, 1947.
2. Editorial: Reactions Following Spinal Punctures, J. A. M. A. **123**: 355, 1943.
3. Peluse, S.: Postspinal Anesthetic Headache, Illinois M. J. **67**: 377, 1935.
4. Dattner, B.: Management of Neurosyphilis, New York, 1944, Grune & Stratton, Inc.
5. Jennings, W. K., and Karabin, J. E.: Incidence of Headache, Nausea and Vomiting Following Spinal Anesthesia, Am. J. Surg. **46**: 317, 1939.
6. Kunkle, E. C., Ray, B. S., and Wolff, H. G.: Experimental Studies on Headache, Arch. Neurol. & Psychiat. **49**: 323, 1943.
7. Watson, T. P.: Postspinal Anesthetic Headache, Canad. M. A. J. **49**: 121, 1943.

ACANTHOSIS NIGRICANS ASSOCIATED WITH CANCER OF THE LUNG

Report of a Case

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THE association between acanthosis nigricans and visceral carcinoma is too well known to require repetition. However, the rare occurrence of involvement of the respiratory tract by the malignant process seems sufficient reason to warrant reporting a case. Curth,[1] in 1943, reviewed the literature very thoroughly and found 384 reported cases of acanthosis nigricans of which 191 were associated with cancer of an internal organ and 193 were of the so-called benign type in which only the skin lesion was present. In the former group she found three cases of cancer of the lung and of these only two were proved by post-mortem examination. Of the 191 cases associated with cancer, the tumor was abdominal in 92.67 per cent while the breast was the site of the malignancy in 4.7 per cent. In one case mediastinal cancer was suspected but not proved. Curth's review gives a very complete bibliography of the literature up to 1943. Since that date there have been no additional cases reported of cancer of the lung in association with acanthosis nigricans.

CASE REPORT

H. D., a 50 year-old white man, was admitted to the Veterans Administration Hospital, Manhattan Beach, April 8, 1948, complaining of cough and expectoration and shortness of breath of four weeks' duration. He had a chronic cough during the past eighteen years and, during November, 1945, had had lobar pneumonia from which he never made a complete recovery. In March, 1948, he developed sharp pain across the anterior portion of the midchest, shortness of breath on exertion, increase in cough, especially at night. He developed a low-grade fever and was said to have fluid in the chest. Since February, 1948, he had noted that the skin in the axillae, nipples, and around the base of the neck had become increasingly dark. There had been a loss of forty pounds since February, 1948.

The family history was noncontributory. There were nine siblings, none of whom were known to have had skin changes or malignant disease. His father died of arteriosclerotic heart disease. His mother was living and well.

The past history revealed he had a skin lesion on the right side of the face for which he received injections of "gold and liver" in 1943. The nature of this lesion was not known.

Physical Examination.—On admission, temperature was 101.4° F., the pulse 104, the respirations 28, and the blood pressure 92/60. He was a well-developed and moderately nourished white man appearing chronically ill and in mild respiratory distress. Surrounding the

From the Medical Service, Veterans Administration Hospital, Manhattan Beach.

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nipples, in the axillae, at the base of the neck, particularly posteriorly, and between the digits of both hands, there were marked deposits of dark brown pigment. In the axillae and about the base of the neck, the skin was velvety in appearance with increase in thickness. The nasal septum was deflected to the right and the nasal mucosa was congested. There were many missing teeth and considerable caries of those remaining. From the angle of the right scapula to the base of the lung there was dullness to percussion with numerous medium moist râles but no change in breath sounds. There was no abnormality noted in the cardiovascular system. There were well-healed scars in the right inguinal region and the right lower quadrant at the sites of previous herniorrhaphy and appendectomy, respectively.

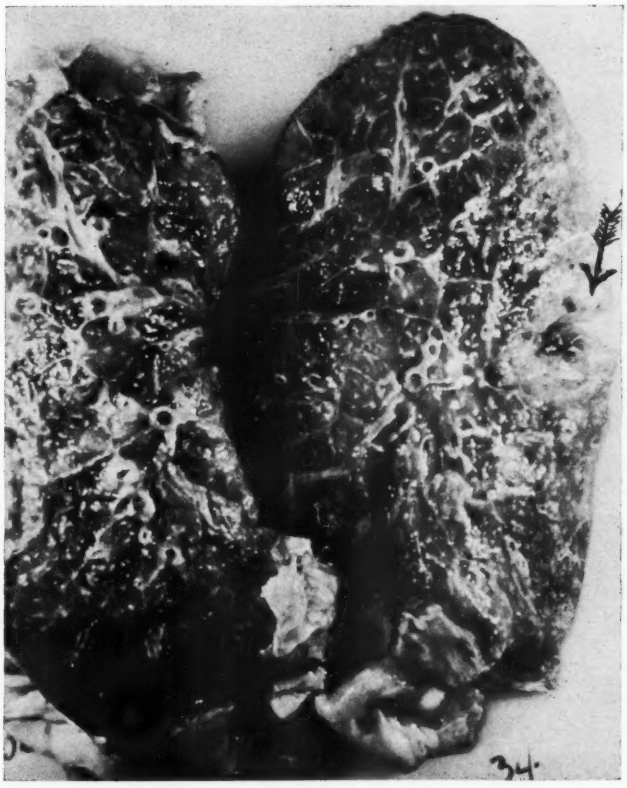

Fig. 1.—Right lung showing primary tumor mass and secondary infiltration.

Laboratory Findings.—The hemoglobin was 12.5 Gm., the red blood cell count 4,400,000, the white blood cell count 13,500, 83 per cent neutrophils, 12 per cent lymphocytes, 4 per cent monocytes, 1 per cent eosinophils, sedimentation rate 28 (Wintrobe); urinalysis negative; Kahn negative. Sputum was examined repeatedly for tubercle bacilli and none were found. Blood culture was negative. X-ray examination of the chest showed a thick-walled abscess, probably carcinomatous, at the apex of the right lower lobe. There was some pneumonic infiltration of the left lower lung and bilateral pleuritic infiltration adjacent to the left diaphragm. Electrocardiogram showed auricular fibrillation on one occasion and normal sinus rhythm on another and there were evidences of myocardial damage as well.

Fig. 2.—Low-power section of involved skin showing papillary hyperkeratosis and increase in prickle cell layer (acanthosis).

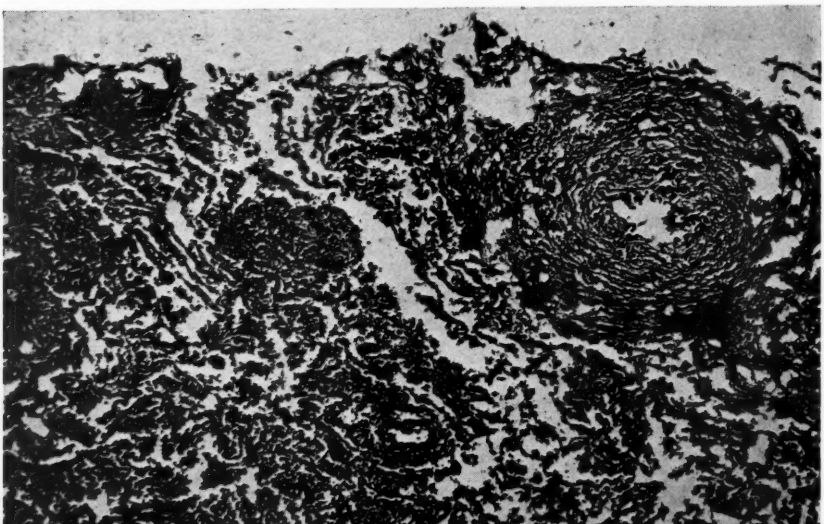

Fig. 3.—Low-power section (×80) of lung showing invasion by tumor tissue.

Course in Hospital.—During the period of hospitalization, he ran a septic temperature, ranging between 99° and 103° F. He received penicillin without effect. Bronchoscopy was done in an attempt to find the site of the tumor, but only large quantities of thick tenacious sputum were seen in the right bronchus. Dyspnea became progressively worse and in spite of supportive therapy, he died on April 26, 1948.

Post-mortem Examination.—Autopsy was performed* and the examination revealed fibrous adhesions in both pleural cavities with about 200 c.c. of slightly cloudy amber fluid. The pericardial sac was obliterated by fibrous adhesions. The lower lobe of the right lung was partially adherent to the pericardium, which in turn was adherent to the diaphragm. There were discrete, firm, lymph nodes in the superior mediastinum which were creamy white in color and cheesy in consistency. Many of the lymph nodes contained anthracotic pigment. The left lung was smaller than usual and boggy. On section, it was edematous and contained

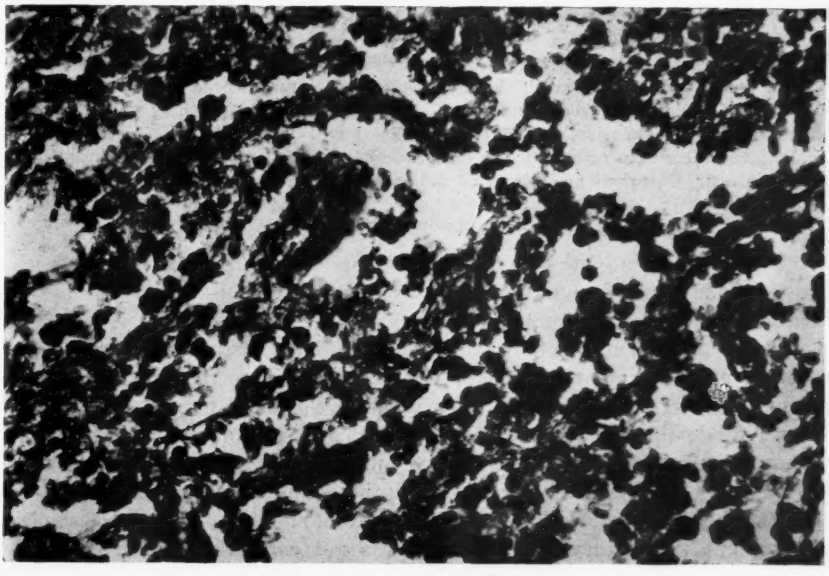

Fig. 4.—High power (×450) of same portion of lung showing anaplastic cancer cells with hyperchromatic nuclei.

atelectatic and fibrotic areas. The bronchioles were dilated and the walls roughened and thickened. The lumen was filled with thick tenacious blood-tinged exudate. The right lung filled the entire pleural cavity and weighed 1,500 Gm. and was firm in consistency. On section, the upper lobe was firm and almost non-air-containing. The outer one-third of the superior portion of the lower lobe contained a more or less circumscribed grayish-white, friable, firm mass, 4 cm. in diameter with a central cavity 1 cm. in diameter which was filled with a thick green purulent exudate. No bronchiole could be traced in this area (Fig. 1). The remaining portion of the lower lobe was firm, friable, and contained purulent exudate. Careful dissection of the right bronchial tree failed to show any ulceration or tumor. Bronchial lymph nodes averaged 1 to 2 cm. in diameter and were firm in consistency. The heart weighed 800 grams. The pericardium was thickened and measured up to 0.5 cm. in some sections. There was no unusual thickening of the ventricular walls. The coronary arteries were all patent. The esopha-

*By Dr. Lester M. Fox, Chief of Pathological Service at Veterans Administration Hospital, Manhattan Beach.

gus was surrounded by several 1 cm., fairly firm, lymph nodes of cheesy consistency. The mucosa of the esophagus, stomach, and small and large intestines was free of any ulceration or neoplasm. The kidneys were normal.

Microscopic Examination.—There was papillary hyperkeratosis of the skin and formation of epithelial papillae with long, thin, rete pegs. The prickle cell layer was moderately thickened. Some of the basal cells contained melanin (Fig. 2). The involved portion of lung showed marked distortion of the architecture, thickening of the pleura, and invasion by tumor cells. Lung tissue was invaded by masses of these cells which were large, round, polygonal, hyperchromatic, and in some areas, giant cell in type. The nuclei were large, almost filling the cell, and having only a small rim of cytoplasm. There was no definite grouping or distinguishing features. The interstitial tissue was also invaded by tumor cells following the aforementioned pattern. Many of the alveoli were filled with sheets of tumor cells, giving the appearance of a pneumonitis. The linings of lymph vessels were replaced by tumor cells and in many places the lumens and walls of the lymphatics were filled with tumor tissue. The adjacent alveoli were collapsed and hemorrhagic (Figs. 3 and 4). The lymph nodes showed distortion of architecture with complete invasion by hyperchromatic, bizarre-shaped, large, round, and polygonal cells, some of which were multinucleated. In some sections, the capsule of the gland was thickened and much of the tumor tissue was necrotic. There was marked thickening of the visceral pericardium with vascularization and fibrosis. Focal areas of round and plasma cells still persisted and there were nests of tumor cells, some of which were found in the lymphatics of the epicardium. There was no evidence of metastasis to other organs.

DISCUSSION

The significance of the association between a skin lesion in itself benign and cancer has been discussed recently by Curth,[2] who pointed out the various theories which have been propounded to explain this association. It was long thought that pressure from the cancerous lesions in the abdomen on the adrenals or the sympathetic chain was responsible for the pigmentary cutaneous changes. However, the occurrence of the small percentage of cases of extra-abdominal malignancy disproves this theory and, where tests for adrenal insufficiency have been carried out, they have been uniformly negative. Genetic relationships between acanthosis and cancer seem to exist. A study of the case reported here, however, reveals no familial history of similar disease, but the history of collaterals was not carefully investigated and, therefore, no real conclusions can be drawn.

The possible influence of steroid hormones[3] on the development of acanthosis has been suggested because of the appearance of the lesion with the onset of menstruation in at least two members in one family. In our case, no such influence was suggested either by the history or physical findings.

Whatever the fundamental reasons may be, it seems apparent that there must be a close etiological connection between the skin lesion and the visceral neoplasm. It is a curious and certainly provocative coincidence that a characteristic and unmistakable external lesion is in 50 per cent of the cases associated with an internal malignant tumor. Further studies are certainly needed to clarify this relationship and, perhaps, to give an answer to the problem of what initiates the cancerous growth in one group of cases but is lacking in the other. The internist or dermatologist who first sees and recognizes acanthosis nigricans has a responsibility to solve this perplexing riddle.

SUMMARY

A case of acanthosis nigricans occurring in a 50-year-old white man in association with anaplastic carcinoma of the lung is reported. This is the third proved case of carcinoma of the lung to be reported as occurring with acanthosis nigricans. The possible etiological factors are briefly discussed.

REFERENCES

1. Curth, Helen Ollendorff: Cancer Associated With Acanthosis Nigricans: Review of Literature and Report of a Case of Acanthosis Nigricans With Cancer of the Breast, Arch. Surg. **47**: 517-522, 1943.
2. Curth, Helen Ollendorff: Acanthosis Nigricans and Its Association With Cancer, Arch. Dermat & Syph. **57**: 158-170, 1948.
3. Jadassohn, W.: Acanthosis Nigricans kombiniert mit Stoffwechselstörung (pathologische Fettsuch), Zentralbl. f. Haut- u. Geschlechtskr. **21**: 43, 1927. Cited by Curth.

A REPORT OF A CASE OF LOCALIZED BRONCHOPULMONARY ASPERGILLOSIS SUCCESSFULLY TREATED BY SURGERY

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BRONCHOPULMONARY aspergillosis was first described by Virchow in 1856. Toward the end of the nineteenth century the disease was reported with increased frequency among pigeon feeders and hair manipulators in France. The organism was present in the grain which the pigeon stuffers masticated and then fed directly into the mouths of the birds. In the case of the wig makers, corn meal was used to degrease the hair and the men frequently worked in an atmosphere heavy with its dust. It was thus considered a primary occupational disease, which was also known to occur among other grain handlers, such as threshers and fur cleaners who use rye flour for degreasing.[1] Chickens, turkeys, and other birds commonly acquire the disease after contact with moldy feed.

In the United States several clinical types of pulmonary aspergillosis have been described. The mildest form has been characterized by an acute tracheobronchitis, which usually runs a benign course.[2, 3] Cawley[4] and Gerstl and associates[5] described a severe pneumonic type which resulted in generalized aspergillosis and death. Chronic forms have also been described showing marked resemblance to tuberculosis[6] or neoplasm. Gerstl was the first to report a successful lobectomy for localized bronchopulmonary aspergillosis. This paper presents a report of the second case which resembled a neoplasm radiologically and which was successfully extirpated.

CASE REPORT

A 35-year-old, well-developed, white man was admitted to the hospital on May 2, 1949, with no complaints. Prior to induction into the Army in August, 1941, he was a salesman in a chicken-feed store. Subsequent to separation in August, 1945, he was actively engaged in chicken farming. According to the patient, a "spot" was seen on the x-ray film of his chest taken by a mobile unit two weeks before admission. He was referred to a near-by hospital where studies of the sputum for tumor cells and tubercle bacilli were negative. Bronchoscopic examination showed a normal tracheobronchial tree. He was then transferred to this hospital.

Past history revealed that he had had a bout of left lower chest pain with fever and a nonproductive cough in 1947, two weeks following a dental extraction. Four months later he had a severe cold with a hacking cough, productive of a small amount of yellowish sputum with occasional blood-streaking. These symptoms cleared after taking a sulfonamide drug. He was well until three months before admisssion when he developed another severe cold with the expectoration of a small amount of nonbloody sputum.

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Physical examination revealed a well-nourished patient who did not appear ill. Temperature was 98.6° F. and the pulse rate was 84 per minute. Examination of the lungs was negative. X-ray films of the chest revealed an ovoid area of infiltration about 4 cm. in diameter in the apical segment of the left lower lobe. There was a questionable high light in the region of this infiltration (Fig. 1).

Laboratory studies revealed that the red blood cell count was 4,800,000, hemoglobin 14.5 Gm., and the white blood cell count was 10,000. Tuberculin intradermal test (P.P.D.) was positive in the first strength. A coccidioidin skin test (1:1000) was negative. Sedimentation rate was 7 mm. per hour (Wintrobe). Sputum culture showed many streptococci, *Hemophilus influenzae*, diphtheroids, and staphylococci. Six concentrated specimens of the gastric juice were negative for tuberculosis and pathogenic fungi.

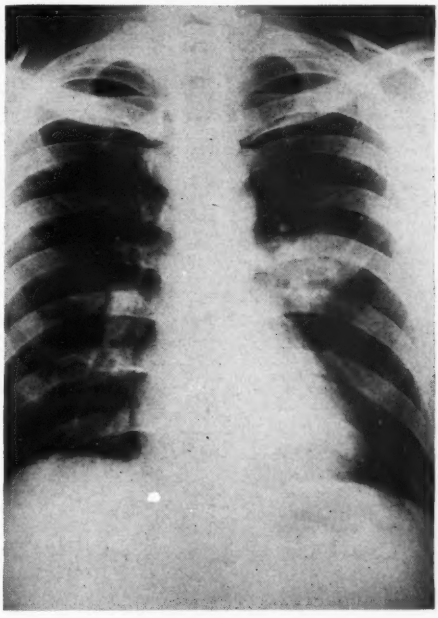

Fig. 1.—Anteroposterior view of lung reveals an ovoid area of infiltration in the apical division of the left lower lobe.

Clinical Course.—The patient remained asymptomatic and afebrile. Bronchoscopy again revealed a normal tracheobronchial tree. The differential diagnoses were chronic lung abscess, tuberculoma, and circumscribed carcinoma of the lung. On May 25, 1949, under endotracheal anesthesia the pleura was opened through the bed of the seventh rib. The lesion in the apical segment of the lower lobe was rounded and moderately firm and encroached upon the origin of the basilar bronchus. The fissure was developed with difficulty because of numerous vascular adhesions. The left lower lobe was removed by the technique of individual ligation of the vessels and the bronchus. A No. 26 catheter was inserted in the eighth interspace and the wound was closed in layers with silk. The patient was discharged on the ninth postoperative day after an uneventful recovery. He was completely asymptomatic when seen seven months later.

Pathology Report.—The specimen consisted of a pulmonary left lower lobe, measuring 17 by 17 by 2½ cm. and weighing 210 grams. A few fibrous tags were attached to the apex. The superior portion of the apical segment felt indurated and nodular. At a point 1½ cm.

from its origin, the apical bronchus opened into an irregular, partially loculated cavity, measuring 4 by 2 by 2½ cm. A branch of the apical bronchus also communicated separately with the cavity. The cavity was lined by a pink-tan, opalescent, smooth, glistening membrane, which was very easily separated from the cavity wall (Fig. 2). The wall was 2 mm. thick

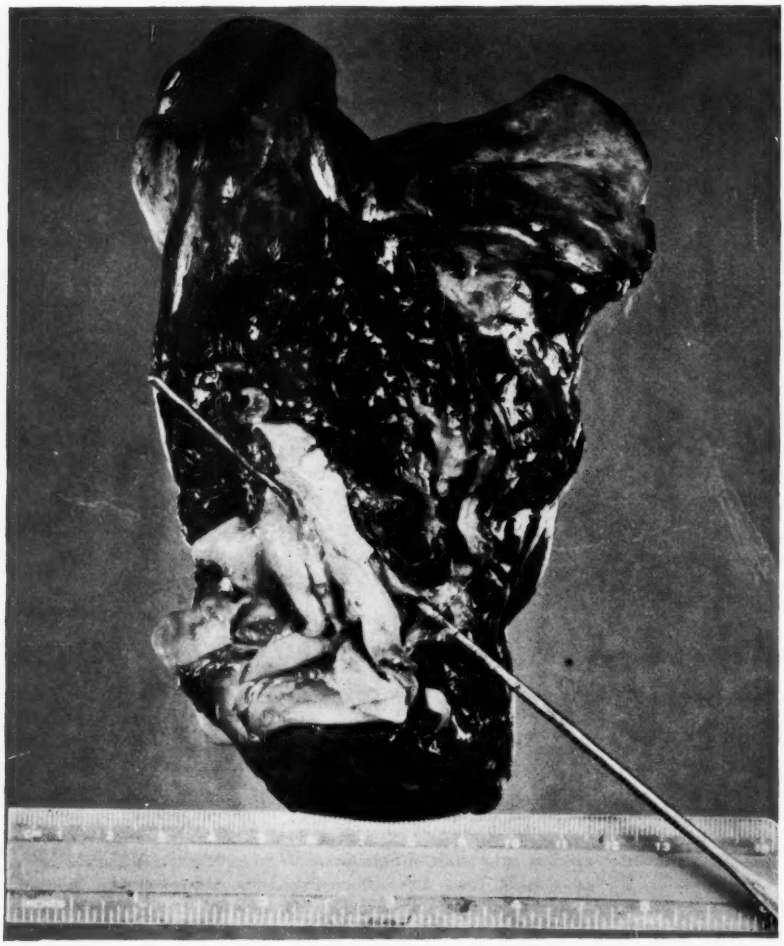

Fig. 2.—Lung specimen showing two bronchi entering the cavity which is filled with a lightly attached, glistening, opaque aspergillosis membrane.

and smooth, except for a single, flat, irregular projection 1 cm. in diameter, which consisted of incompletely removed membrane. The parenchyma surrounding the cavity was indurated. On microscopic examination, the wall of the cavity was lined partially by pseudostratified ciliated columnar epithelium and partially by metaplastic squamous epithelium. There were also transitional ulcerated areas and re-epithelialized ulcerations covered by a single layer of cuboidal cells. Mucous glands, smooth muscle, and cartilaginous plates were found within the wall of the cavity which was fibrotic and heavily infiltrated with plasma cells, mac-

rophages, eosinophiles, and polymorphonuclear leucocytes. The surrounding lung parenchyma was also chronically inflamed. The lymphocytic infiltration was organized in some areas into follicles, some of which contained germinal centers. The lining mucosa of the cavity was

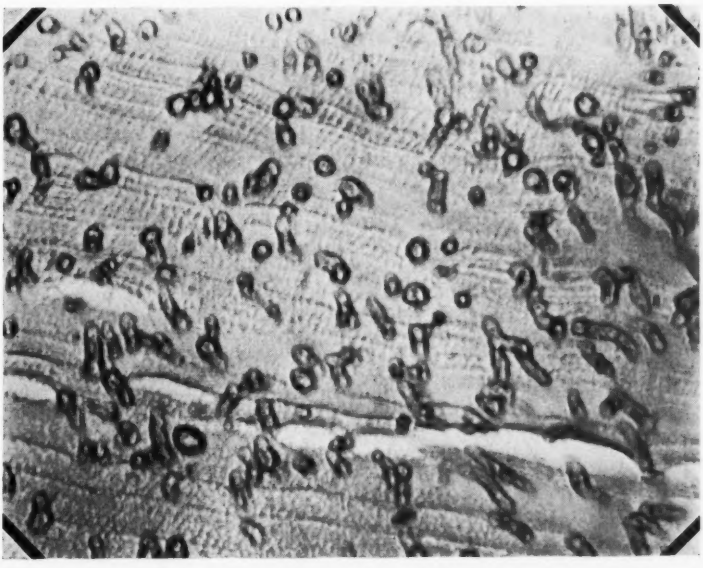

Fig. 3.—Oil immersion photomicrograph of mycelial membrane shown in Fig. 2.

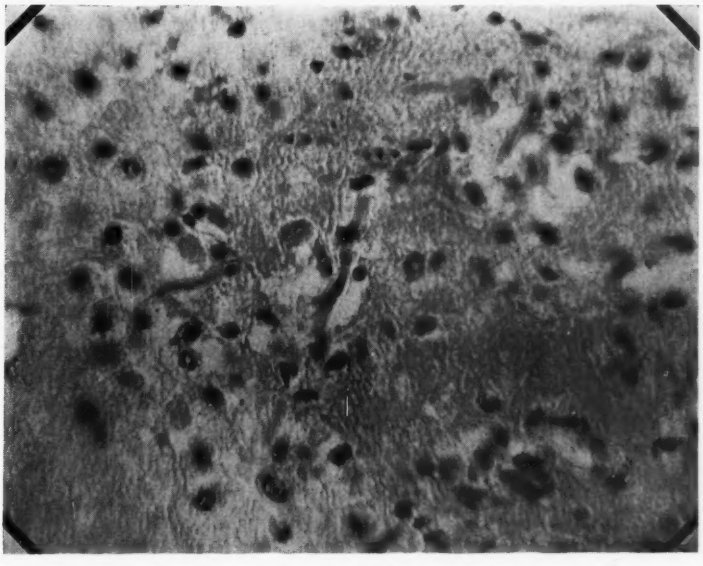

Fig. 4.—Oil immersion photomicrograph of thrombus containing mycelia in portal vein radicle of rabbit, produced by intravenous injection of suspension of aspergillus grown from membrane of patient's cavity.

frequently invaded by polymorphonuclear leucocytes. Granulation tissue was everywhere evident beneath the thickened basement membrane and fresh thrombi were attached to the ulcerated foci of the mucosa. Section through the lining membrane revealed a hyaline laminated structure, throughout which were present the branching mycelia of a fungus, frequently having the appearance of spores on cross-section (Fig. 3). On culture this membrane yielded a pure growth of colonies characteristic of aspergillus.* On June 19, 1949, a heavy saline suspension of the organisms was injected into the ear vein of a rabbit, which died in about forty-eight hours. Sections through the rabbit's lungs revealed marked congestion and edema and numerous small granulomas consisting of macrophages filled with small, round, foreign bodies consistent with spores. Some of these granulomas also showed foci of necrosis and polymorphonuclear leucocytic infiltration. Numerous fungal mycelia were present throughout the sinusoids of the liver, in which there were numerous foci of necrosis and thrombosed veins. Fungal mycelia were seen within the thrombi (Fig. 4). The most luxurious growth was found in the kidneys where large areas of necrosis occurred in conjunction with the growth and where blood vessels invaded by the fungi were also thrombosed. No fungi were noted in the spleen.

COMMENT

The gross and microscopic picture of the lesion, the demonstration of aspergillosis in the membrane lining the cavity, a positive virulence test in an inoculated rabbit, and the failure to demonstrate any other causative agent, are the reasons for considering this a case of primary bronchopulmonary aspergillosis. In addition, the patient was exposed to moldy feed in his occupation. Preexisting bronchiectasis is unlikely because more than one bronchus entered the cavity, as in Deve's[7] case, and the lesion was confined to the apical segment of the lower lobe, an unusual site for localized bronchiectasis. The possibility exists, however, that the aspergillosis was superimposed on a chronic lung abscess, secondary to the dental extraction of 1947.

Iodides and thymol have been efficacious in the treatment of the tracheobronchitic and tubercular forms of the disease. Surgical intervention should be employed as an effective measure in the treatment of localized bronchopulmonary aspergillosis.

SUMMARY

The third case of localized bronchopulmonary aspergillosis successfully treated by surgery has been reported. The patient has shown no clinical or x-ray evidence of recurrence of disease following operation.

REFERENCES

1. Coe, G. C.: Primary Bronchopulmonary Aspergillosis, An Occupational Disease, Ann. Int. Med. **23**: 423-425, 1925.
2. Coon, E. H., Smith, H. B., and Walsh, J. C.: *Aspergillus fumigatus* Infection of Tracheobronchial Tree; Case, M. Times, New York **74**: 225, 1946.
3. Van Ordstrand, H. Scott: Pulmonary Aspergillosis; With Report of a Case, Cleveland Clin. Quart. **7**: 66-73, 1940.
4. Cawley, E. P.: Aspergiliosis and the Aspergilli. Report of a Unique Case of the Disease, Arch. Int. Med. **80**: 423-424, 1947.
5. Gerstl, B., Weidman, W. H., and Newmann, A. V.: Pulmonary Aspergillosis, Report of Two Cases, Ann. Int. Med. **28**: 662-671, 1948.
6. Donaldson, J. M., Jr., Koerth, C. J., and McCorkle, R. G.: Pulmonary Aspergillosis, J. Lab. & Clin. Med. **27**: 740-746, 1942.
7. Deve, F.: Une nouvelle forme á natomo-radiologique de mycose pulmonaire primitive, le mega-mycetoma intrabronchectasique, Arch. méd.-chir. de l'app. respir. **13**: 337-361, 1938.

*The fungus was identified as *Aspergillus fumigatus fresenius* by Dorothy I. Fennell of the Fermentation Division, Northern Regional Research Laboratory, Peoria, Ill.

NEUROFIBROSARCOMA OF THE ANTERIOR MEDIASTINUM

Surgical Removal

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IN A comprehensive review of the literature on "Mediastinal Tumors and Cysts" Thompson[1] found only two tumors of neurogenic origin arising in the anterior mediastinum. On the basis of observations in 135 mediastinal neurogenic tumors Blades[2] concluded that these tumors are almost always located in the posterior mediastinum. His collected series of cases consisted of 70 ganglioneuromas, 24 fibroblastomas, 22 neurofibromas, and 19 neurinomas.

The case to be presented in this paper is one of a neurofibroma which had undergone sarcomatous changes. It was located in the anterior superior mediastinum and was large enough to cause such critical tracheal compression that its removal was performed as an emergency procedure. Because of the rarity of this pathologic entity in the anterior mediastinum and the successful end result we feel that this case is worthy of report.

CASE REPORT

T. S., a 63-year-old gardener, entered Kings County Hospital on Jan. 4, 1947, with a ten-day history of dyspnea made worse three days before admission by an upper respiratory infection. Past history was noncontributory.

On admission to the hospital the patient was acutely sick, orthopneic, dyspneic, and cyanotic from the crown of the head down to the middle of the chest. The superficial veins in the neck and on the chest and abdomen were quite prominent. The pupils were dilated and reacted to light and accommodation. The right lobe of the thyroid appeared enlarged. There was fullness in the suprasternal notch and deviation of the trachea to the right. The anterior superior mediastinal region was widened to percussion for about one inch to either side of the sternum. The blood pressure in the right arm was 148/98 and 158/90 in the left arm. The remainder of the physical examination was essentially negative.

Radiographic examinations of the chest (Fig. 1) which included kymography and barium sulfate ingestion studies revealed a large anterior superior mediastinal mass which extended to both sides of the midline and upward into the neck. The lateral borders of the opacity were sharply demarcated from the surrounding tissues. The edges of the tumor were smooth and regular. There was considerable displacement of the trachea to the right. Kymographic studies did not show any pulsations within the mass. Fluoroscopy during barium sulfate ingestion showed the esophagus to be deviated to the right in the region of the tumor.

On admission to the hospital the patient was immediately placed in an oxygen tent. His condition improved during the next few days so as to permit investigation of the cause of his illness. The improvement, however, was not long lasting. On Jan. 22, 1947, he began to have respiratory distress even in the oxygen tent. The following day there was increased dyspnea and cyanosis. It was, therefore, deemed advisable to remove the tumor compressing the trachea as soon as possible. In view of the fact that the clinical diagnosis rested between (1) retrosternal thyroid and (2) primary anterior superior mediastinal tumor, it was felt

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that the fastest and least traumatic approach to the tumor would be through the neck. Accordingly, on Jan. 23, 1947, under endotracheal anesthesia, a collar incision was made at the base of the neck. After the thyroid gland had been exposed, a discrete nodule was found within the substance of the lower pole of the right gland. It was then noted that the tumor

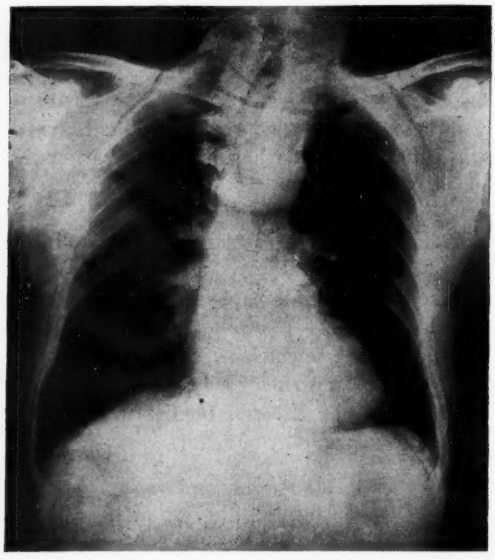

Fig. 1.—Roentgenogram of the chest taken on admission to the hospital. There is a large superior mediastinal shadow with deviation of the trachea to the right.

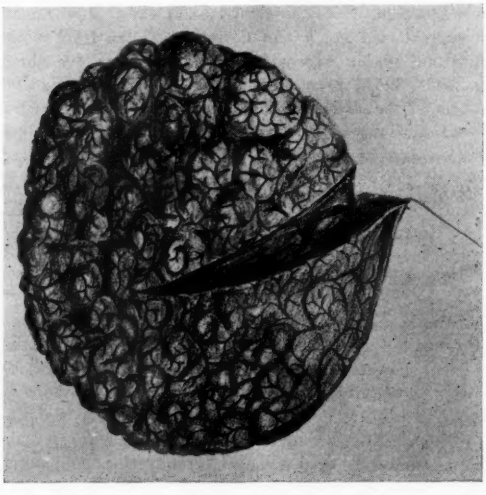

Fig. 2.—Drawing of the tumor removed from the anterior superior mediastinum. The tumor measured approximately four inches in diameter.

Fig. 3.

Fig. 4.

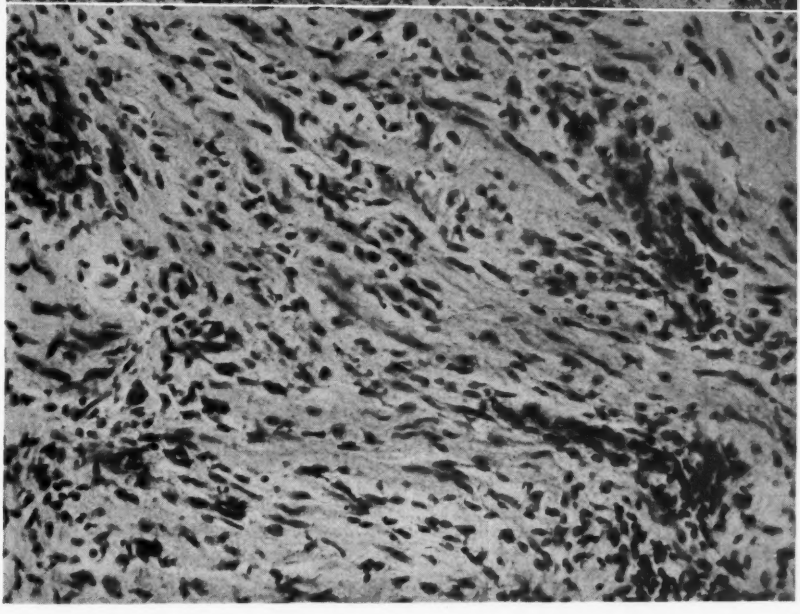

Fig. 3.—Photomicrograph of the tumor (magnification ×100) showing cytology of the tumor.

Fig. 4.—Photomicrograph of the tumor (magnification ×225) showing the deeply staining hyperchromatic markedly fasciculated structure of a neurofibrosarcoma.

in the mediastinum was *not* connected to the thyroid gland. The nodule within the thyroid gland was removed. Traction upon the mediastinal tumor in an attempt to deliver it into the neck produced marked respiratory distress, cyanosis, and elevation of the pulse rate. In order to decompress the mediastinum and relieve the pressure upon the trachea, the neck wound was abandoned and an incision made on the left side of the chest over the second rib anteriorly. About three inches of the cartilage and bone were removed. The tumor bulged through this opening and the patient's breasing imprvoed, the cyanosis diminished, and the pulse rate declined. After the patient received about ten minutes rest, the operation was resumed and the tumor removed through an opening in the left mediastinal reflection of pleura. The chest wall wound and the one in the neck were closed by coaptating the corresponding layers of tissue. The patient's immediate postoperative condition was fair. After the third postoperative day he showed steady and progressive improvement except for the development of an ischiorectal abscess which required incision and drainage on the sixth postoperative day.

Fig. 5.—Roentgenogram of the chest taken two years after the removal of the mediastinal tumor.

Microscopy of the nodule removed from the thyroid gland showed adenomatous changes.

The tumor mass removed from the retrosternal region was described as follows: The mass was ovoid in shape and measured 4 by 3 inches in diameter (Fig. 2). Microscopy showed the presence of collagenous fibrous tissue with fasciculations. Many of the cells were elongated, large, and oval in shape with eccentric hyperchromatic large reticular nuclei. In some areas lymphocytic infiltration and calcification were noted (Figs. 3 and 4). The diagnosis was mediastinal neurofibrosarcoma.

The patient was discharged from the hospital two months after the operation on the chest. He was seen periodically in the Chest Clinic of this hospital, the last time being on Jan. 10, 1949 (Fig. 5). At that time he stated that he had gained considerable weight, that he had had no recurrence of symptoms or pain in the chest and that he had been

working as a gardener and handyman ever since his discharge from the hospital. Because of his employment by the Kings County Hospital he has been seen frequently, outside of the clinic, and has remained well to date.

DISCUSSION

Heuer and Andrus[3] demonstrated that intrathoracic tumors of nerve tissue origin may arise from any of the nerve elements as well as from their sheaths or connective tissue contents. Another possible source of origin of these tumors is cell rests deposited ectopically during embryonic development which become active during later life. A review of the cases of neurofibromas and neurofibrosarcomas reported in the literature shows that they usually occur singly in the chest and probably arise from the connective tissue elements of nerve. In a review of 105 cases of intrathoracic neurogenic tumors Kent, Blades, Valle, and Graham[4] observed that 39 (37 per cent) were malignant. They also noted that neurogenic tumors arising within the mediastinum were less liable to malignancy than those originating in the more peripheral parts of the thorax. The average age of the patient in 55 cases of benign neurogenic tumor was approximately 26 years and in 15 cases of malignant tumors it was 17 years. Our patient did not conform with the observations of other writers either in location of the tumor or age of the patient.

The symptom complex in cases of neurogenic mediastinal tumors may take one of three forms—(1) neurologic, (2) manifestations of increased mediastinal pressure, and (3) pulmonary—one of which predominates. Not infrequently there is a combination of symptoms. Most writers believe that the onset of symptoms is chiefly neurologic and that encroachment upon the mediastinum and lung occurs late in the course of the expansion of the tumor. Harrington called attention to the fact that benign tumors are usually asymptomatic even when of considerable size, whereas severe intercostal pain was a common feature in malignant neurogenic tumors and in benign tumors undergoing malignant degeneration. All symptoms and signs of nerve involvement were absent in our patient. Interference with respiration due to pressure upon the trachea was the chief reason for considering the tumor preoperatively to be a retrosternal thyroid goiter. The bizarre symptomatology in our case was due to the unusual location of the neoplasm, that is, in the anterior superior mediastinum.

The roentgenologic findings in neurogenic mediastinal tumors usually consist of an area of mediastinal opacity the borders of which are smooth and more rarely lobulated. Occasionally there may be noted erosion of the osseous tissue in juxtaposition to the tumor. This is due to local pressure and does not indicate malignant transformation. In the lateral roentgenogram these tumors are usually found in the posterior mediastinum. In our case the tumor was located in the anterior mediastinum. Neither benign nor malignant tumors of neurogenic origin respond to x-ray therapy. The so-called "test dose" of deep roentgen rays is not warranted in mediastinal neurogenic tumors. The treatment of such tumors is surgical. The recent advances in thoracic surgery and anesthesiology have made exploratory thoracotomy a

safe procedure. At the time of such intervention not only may the surgeon remove tissue for biopsy purposes but he may also remove the lesion, eradicating the disease.

RÉSUMÉ

A case is presented (with a three-year follow-up) in which a primary neurofibrosarcoma was removed from the anterior superior mediastinum. A brief discussion of mediastinal neurogenic tumors is also presented.

REFERENCES

1. Thompson, J. V.: Mediastinal Tumors and Cysts, Internat. Abst. Surg. **84:** 195-221, 1947.
2. Blades, B.: Relative Frequency and Site of Predilection of Intrathoracic Tumors, Am. J. Surg. **54:** 139-148, 1941.
3. Heuer, G. J., and Andrus, W. DeW.: The Surgery of Mediastinal Tumors, Am. J. Surg. **50:** 143-224, 1940.
4. Kent, E. M., Blades, B., Valle, A. R., and Graham, E. A.: Intrathoracic Neurogenic Tumors, J. THORACIC SURG. **13:** 116-161, 1944.
5. Harrington, S. W.: Surgical Treatment in Fourteen Cases of Mediastinal or Intrathoracic Perineurial Fibroblastoma, J. THORACIC SURG. **3:** 590-611, 1934.

CAVERNOUS HEMANGIOMA OF THE MEDIASTINUM

Case Report

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BECAUSE of the relative rarity of mediastinal tumors of blood vascular origin, such a case is believed suitable for recording.

A recent article from the Mayo Clinic* indicates that an extensive search of the literature on the subject reveals 14 acceptable cases of tumors of the mediastinum which are believed to be of blood vascular origin. The authors added to this series 3 more such cases, making a total of 17 authenticated cases of blood vascular tumors of the mediastinum. There was evidence of malignancy in 8 of the 14 cases gleaned from the literature and 2 of the 3 cases reported in the article showed evidence of malignancy. In the present case report the tumor showed no evidence of malignancy. The 3 cases at the Mayo Clinic occurred among approximately 200 patients with mediastinal tumors operated upon. The relative rarity of such tumors may be understood from this report.

Following is a case report of a similar tumor:

History.—Mrs. C. B., female, aged 18 years, presented herself for examination July 7, 1949. Patient stated that in 1939 she was treated for lobar pneumonia and responded well to one of the sulfonamides. An x-ray examination of the chest was made at that time, but it did not help because when it was taken it showed a homogeneous density obscuring the lower two-thirds of the left lung field, and if any tumor was present at that time it was well hidden by the pneumonic process. Otherwise, the history was noncontributory, consisting of the usual childhood diseases without any sequelae. In February, 1949, the patient was walking down the street in her home city when she noticed a mobile unit of the National Tuberculosis Association and had an x-ray examination made. She had no symptoms at that time that might be related to chest pathology. The physician in charge of the unit did not tell her what he had found but advised her to report to her private physician for examination and repeat x-ray examination. This was done and the examining physician found evidence of tumor which caused him to refer the patient for further examination and treatment. Following the original detection of the tumor, the patient in retrospect had the following complaints: vague distress in the left chest which came and went, bearing no relation to effort. No history of cough, hemoptysis, or weight loss. No dyspnea but fatigued easily. She had particularly noticed that she was unable to swim as well as she could several years ago because of the progressive shortness of breath.

Physical examination revealed no demonstrable abnormalities on careful examination of the entire chest. Blood pressure 120/78, pulse 74 per minute. Heart sounds entirely normal.

X-ray Examination, July 7, 1949.—Posteroanterior and lateral projections of the chest showed the right side essentially clear. The left side revealed a rounded mass extending from the hilum into the lung structure in the midportion of the lung field. Vertical diameter of the shadow was about 11 cm. Lateral borders were smooth in outline. Lateral view showed this mass to be located anteriorly and superimposed on the heart shadow. This tumor was interpreted as being a teratoid type of tumor in the left chest.

From the Thomas-Davis Clinic.
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*Seybold, W. D., McDonald, J. R., Clagett, O. T., and Harrington, S. W.: J. Thoracic Surg. 18: 503, 1949.

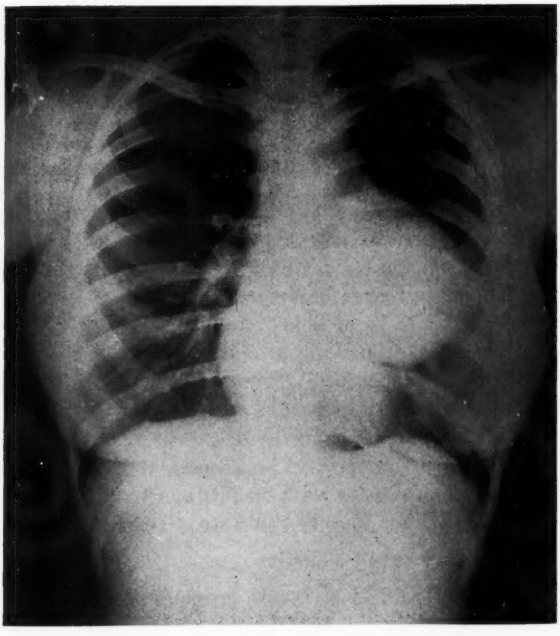

Fig. 1.—Posteroanterior chest preoperatively.

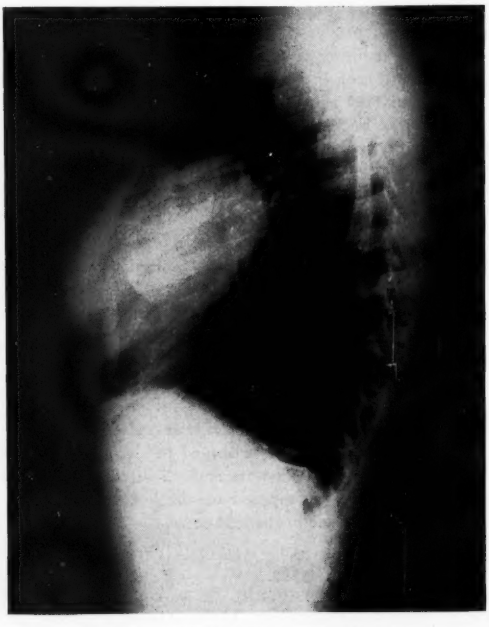

Fig. 2.—Lateral chest preoperatively.

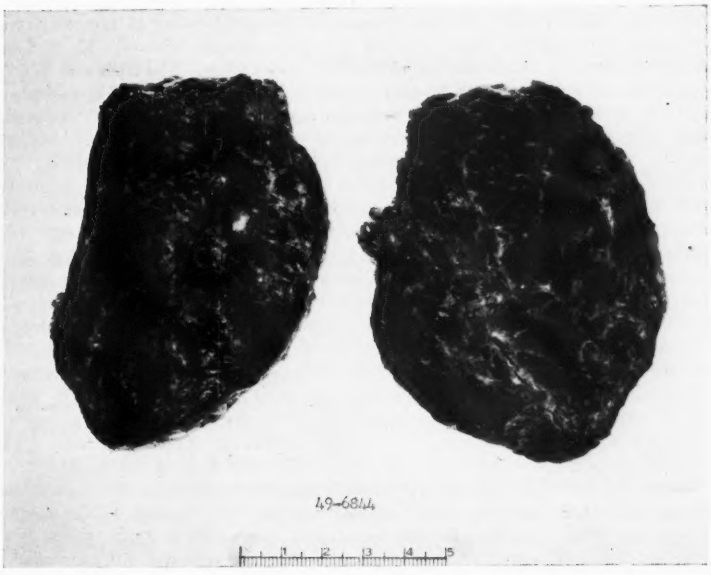

Fig. 3.—Description of illustration is contained within the text.

Fig. 4.—Description of illustration is contained within the text.

Operation, July 13, 1949.—Patient was subjected to exploratory thoracotomy on the left side, and after the lung had been retracted a large mass approximately 10 by 8 by 7 cm. in diameter was located on the left surface of the pericardium. The mass was not attached to the pericardium but seemed to be surrounded by its own capsule. The capsule was incised and the mass was dissected free from its capsular attachment with some difficulty. Dissection progressed to a pedicle approximately 2 cm. in diameter located near the arch of the aorta. The exact origin of the pedicle and the mass was not determined. The mass was removed at its pedicle and hemostasis was secured by suture ligatures. Operation was concluded by closing the thoracotomy incision with closed drainage. Patient made an uneventful recovery and was discharged on the seventh postoperative day.

Pathology Report.—Gross: A somewhat egg-shaped mass which weighed 275 grams, measured roughly 10 by 7.5 by 6.5 cm., and presented a shaggy surface. The mass did not appear to have been readily enucleated. When a section was carried through the mass, a large amount of blood escaped. The substance of the mass had a cavernous structure, blood escaped from the cavernous spaces.

Microscopic: The mass was made up predominantly of blood vascular elements which were often closely packed together. These blood vessels varied markedly in size and in thickness of their walls, the smaller channels were hardly larger than capillary size and possessed walls of capillary thinness. The larger cavernous spaces occasionally possessed relatively thick walls apparently made up chiefly of smooth muscle fibers. The stroma was not richly cellular and was made up of well-differentiated connective tissue, in which there were imbedded rather numerous scattered aggregations of lymphoid cells, apparently inflammatory cells since there was no arrangement of these cells to form typical lymph follicles. No epithelial cells were seen. The various elements were well differentiated. There was no evidence of rapid growth.

Diagnosis.—Large cavernous hemangioma of the mediastinum.

DISCUSSION

A case is presented of a large cavernous hemangioma of the mediastinum occurring in an 18-year-old girl. The symptoms had been so mild as to not cause the patient to seek medical help but accidental discovery and questioning by her local physician brought out mild pressure symptoms dating for approximately one year prior to the time of first examination. The tumor was removed and the patient made an uneventful recovery, leaving the hospital on the seventh postoperative day. Pathologic report on the tumor placed it in a class of mediastinal tumors of blood vascular origin of which relatively few have been reported in the entire literature. Surgery in this case should result in a permanent cure.

SOME ASPECTS OF CAVITY BEHAVIOR IN PULMONARY TUBERCULOSIS

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DESPITE years of close study and therapeutic attacks from many angles, the tuberculous pulmonary cavity continues to be one of the most difficult problems to confront the clinician, by reason of its unpredictable response to treatment.

Of the modern active methods of cavity treatment, namely, collapse therapy, transpleural drainage, and lung resection, the latter is being employed ever more frequently, particularly for those cases which have not responded to the other two less radical forms of therapy. Many cases of advanced bilateral disease, however, are not suitable for lung resection.

Collapse therapy is a well-tried method of treatment which has been attended by a high percentage of successful results. On the other hand, many cases have their chances of survival lessened by very extensive, perhaps overenthusiastic collapse therapy, sacrificing badly needed lung function. This is done in an attempt to force cavity obliteration by extensive irreversible lung collapse through multiple rib resection.

Transpleural cavity drainage is a useful method particularly in conjunction with collapse therapy for large cavities. This procedure, particularly in the closed form, however, is sometimes hazardous to institute because of uncertainty as to the adequacy of pleural space obliteration, and the lack of a sure closed method for bringing about such obliteration.

In the belief that each of the methods mentioned has its place in cavity treatment at the appropriate time, and that the proper method can be best selected for any particular cavity by studying the type of cavity or phase of cavity evolution with which one is dealing, it is suggested that closer attention to cavity behavior than merely the establishment of the existence of a cavity is required for intelligent application of the methods. The need for such cavity study is well recognized. The work of Pearson, Eloesser, Monaldi, Coryllos, Vineberg, Kunstler, and others drew attention to the blocked cavity or "positive pressure cavity," and suggested transpleural drainage for its treatment.

In this article certain aspects of the behavior of cavities, particularly under the conditions of lung collapse and lung expansion, will be discussed. Some experiences with a plan of procedure aimed primarily at the most efficient and lung-sparing use of collapse therapy, but providing at the same time for the safe use of transpleural drainage or the early institution of lung resection, will be described.

Cavity Persistence.—The mechanism of formation of a tuberculous lung cavity is fairly well understood, but the cause or causes of cavity persistence are still debatable. With our present knowledge, one may assume that aside from

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immune-biologic considerations, there are at least two important causes of cavity persistence, of a mechanical nature, which might be called: (1) cavity fixation, and (2) cavity inflation.

By cavity fixation is meant rigidity of the cavity wall due to fibrosis, and also adherence of the surrounding diseased lung to the resistant bony parietes.

By cavity inflation is meant the progressive trapping of air and the products of tissue destruction within the cavity due to a stop-valve effect in the drainage bronchus or bronchi.

Cavity Obliteration.—The mechanism of cavity obliteration, whether spontaneously or under collapse treatment, is not clearly understood. The most direct form of attack on cavity fixation would seem to be collapse therapy by removal of ribs or division of adhesions. Similarly the most direct treatment for cavity inflation should be adequate cavity drainage either in the natural way through the bronchus or by surgical intervention transpleurally.

If the cavity wall and the surrounding lung have become sufficiently rigid with fibrous tissue and calcification, then probably neither collapse nor transpleural drainage, nor any combination of these will cause cavity obliteration. The only effective remedy that one can see for such a cavity is complete eradication by lung resection.

In so far as the mechanism of cavity inflation is concerned, there is no need to postulate hinged flaps or other bizarre trap-door devices in order to produce a stop-valve effect. Partial occlusion of the bronchial lumen by thick secretions on inspiration can easily become complete occlusion on expiration, owing to the enlargement and diminution of the bronchial lumen accompanying inspiration and expiration, respectively. Such things as increased amount of secretion, semisolid or very viscid secretion, narrowing of the bronchial lumen by endobronchial disease, traction on the bronchial wall by fibrous tissue, compression of the bronchus by lung collapse, and increased inspiratory effort as in coughing or physical exertion will clearly favor cavity inflation. Conversely, thinning of the consistency of the secretion or reduction of its amount as after periods of treatment, or reduced inspiratory effort through bed rest, or sedation of useless cough, will lead to diminished cavity inflation, or deflation and occasionally even cavity disappearance.

Hence, it would appear that the phasic character of cavity inflation with periods of increase in size often alternating with periods of decrease in size can be readily explained on the basis of variation in effective bronchial lumen and resulting bronchial drainage.

Cavity Treatment.—While in treatment, cavity inflation is most directly countered by transpleural drainage, and cavity fixation by collapse, it is clear that collapse therapy alone often deals effectively with cavity inflation. Everybody has experience of giant cavities exhibiting obvious signs of inflation including positive pressure and fluid levels, that have gone on to obliteration under collapse therapy without the added assistance of transpleural cavity drainage. This may be at least partly explained by the fact that collapse therapy generally diminishes the amount of air entering the cavity.

This is particularly true of one form of collapse therapy, namely, diaphragmatic paralysis. This procedure puts out of action the bellows effect of the contracting diaphragm, thus reducing the amount of air entering the lung and the cavity at each inspiration. It is possible that this is the most effective factor in diaphragmatic paralysis and not the diminution in lung volume resulting from diaphragmatic ascent. The rapidity with which some lower and midlung cavities disappear after phrenic interruption even with only slight rise of the diaphragm bears out this contention. Inflated cavities exhibiting positive pressures have been seen to disappear after phrenic interruption without transpleural drainage.

In the same way, for upper lung cavities one need not always require drastic reduction in lung volume, with corresponding loss of lung function, in order to secure cavity obliteration. Removal of a few of the upper ribs, or the creation of an air- or fluid-filled space around the upper lung hinders the inspiratory action of the costosternal mechanism and thus diminishes cavity inflation, often resulting in cavity disappearance.

It is unfortunate that the reduction in lung volume caused by collapse means a corresponding reduction in the lumen of the smaller muscle-walled bronchi, and hence interferes with bronchial drainage and favors cavity inflation. In fact, it is quite possible that too great a degree of collapse may in this way, paradoxically, favor cavity persistence. This is seen at times in cases of pneumothorax where all adhesions have been divided and despite optimum concentric collapse the cavity, instead of diminishing, retains its original size or even enlarges. Frequently on permitting the lung to re-expand in such cases, the cavity diminishes and at times disappears (Case 1).

In theory the correct amount of collapse is that which eliminates adherence of the lung to the parietes, giving full scope to the contracting tendency of the fibrous tissue surrounding the cavity, without leading to compression and obstruction of the bronchial lumen. The rapid succession of stages as followed in the classical thoracoplasty, takes little account of this problem of bronchial drainage, and seeks to cause cavity obliteration by sheer reduction of lung volume. The fact that cavities sometimes persist despite extensive rib and transverse process resection, resulting in total lung collapse with corresponding severe and permanent loss of lung function not to speak of physical deformity, raises a question as to the soundness of such a proceeding. Moreover, after a two- or three-stage thoracoplasty, the residual cavity lies so inaccessibly close to the mediastinum that cavity drainage becomes impractical and lung resection remains the only hope. It is felt that the tendency in recent years has been to concentrate too much on the early ridding of the patient of a few tubercle bacilli from the sputum and not enough on preserving adequate lung function so that the patient can carry on a normal existence. To paraphrase Pinner, patients have been cured of their tuberculosis but cannot walk upstairs.

In an attempt to rationalize collapse therapy according to the principles outlined previously, and, above all, scrupulously to preserve lung function, a procedure has been tried over the past four years on a small series of 15 cases,

in 13 of whom the cavity was 5 cm. or over in diameter, and practically all of whom had bilateral disease. Briefly the procedure consists of the following plan:

1. *Anterior thoracoplasty* with resection of practically all of the upper three ribs combined with an extensive *apicolysis* peeling the upper lung down to the level of the lung root. This operation gives the maximum amount of concentric collapse to the cavitated area such as one gets with an ideal selective pneumothorax. The periosteum of ribs one, two, and three together with the intervening intercostal bundles are removed after the ribs have been resected. This facilitates the apicolysis, hinders the reformation of these ribs, and thus permits a long interval between collapse stages. It is believed that anterior thoracoplasty, operating on the supine patient through a short incision, has many advantages over the classical posterior first-stage operation, in bad risk cases, particularly those with large cavities and copious sputum.

2. *A period of re-expansion* of the lung is then allowed over the succeeding weeks, permitting the lung, as it were, to seek its own level of collapse. After the initial collapse the cavity is as a rule still clearly visible though somewhat reduced in size. As the lung then re-expands, the majority of cavities diminish in size instead of enlarging as one might expect. This period of observation is allowed to continue as long as the cavity continues to diminish, over the succeeding eight to twelve weeks or even longer.

Three different types of cavity behavior emerge: (1) One group of cavities go on to complete disappearance, requiring no further interference. (2) A second group having diminished to a fraction of their former size, remain at this stage, showing no further notable variation in size. (3) A third group, having at first diminished in size, reach a stage where they show a marked tendency to re-enlarge.

One can, of course, only theorize as to the cause or causes of this variation in cavity behavior, but it is believed that a satisfactory explanation of the fundamental mechanism can be offered on the basis of the conceptions of cavity fixation and cavity inflation outlined previously, providing a guide to further treatment.

It is suggested that Group 1 cavities are getting adequate bronchial drainage, and the factor of cavity fixation having been overcome by the operation, the cavity proceeds to complete obliteration. Of the sixteen cavities studied, four were of this type; three remained obliterated for from one to four years without further interference. In one case, posterior thoracoplasty was later performed. In a fifth case with bilateral disease, the cavity disappeared but the sputum is still positive.

Group 2 cavities are possibly getting adequate bronchial drainage but they are sufficiently stiff-walled that the factor of cavity fixation has not been overcome by the amount of collapse initially provided, and further compression is indicated in order to obilterate them. This can be provided by rib resection from behind as in the classical thoracoplasty. At this time the two inch-long portions of the third rib, remaining from the anterior operation, are excised together with long lengths of ribs four, five, and, if necessary, six, and the inferior

angle of the scapula is placed inside the next rib. The small inch-long portions of ribs one and two are not disturbed because the lung lies well below their level and will not be influenced by their removal. These rib portions give attachment to the scalene mass—middle and posterior scalenes—which is preserved in an effort to minimize cervicodorsal scoliosis. Of the sixteen cavities studied, eight were of this type. Five of these were obliterated by further collapse and have remained so for periods varying from six months to four years; one patient died after resection of ribs four, five, and six from behind, the cause of death being undetermined; one patient left the hospital against advice, after the anterior operation, and has not been traced; one patient developed a contralateral spread after the anterior operation. Posterior thoracoplasty later failed to obliterate this cavity.

Group 3 cavities are probably true retention or inflated cavities, which are not amenable to collapse therapy. They can now be treated by transpleural drainage or lung resection. If resection cannot be considered because of extensive disease, transpleural drainage can be done more easily at this stage than after further collapse stages have pushed the cavity dangerously close to the mediastinum and reduced it to a size where its localization is difficult. If cavity drainage is decided upon, this can be instituted anteriorly by a modification of the Monaldi method. The pleural space has been obliterated by the initial operation, but a part of the apicolysis space may still remain, and the drainage catheter cannot be passed through this space because of the danger of infection. The surface of the lung is therefore exposed through a short incision before inserting the catheter into the cavity, and if the apicolysis space is still open it is walled off from the catheter entrance with a flap of pectoralis muscle which is readily available. After the cavity has become reduced by drainage, it is probably advisable to follow with further rib resection from behind in order to keep these dangerous retention cavities obliterated. Of the sixteen cavities studied, two were of this type. They have remained obliterated for from three to four years after treatment in the manner outlined.

It should be emphasized that an essential part of the treatment method described is the long observation period of lung re-expansion allowed after the initial collapse operation. This is of value for two reasons: (1) certain cavities go on to obliteration without further interference; (2) information is gained regarding the cavities that do not become obliterated, which acts as a guide in their further treatment.

As with other methods there are certain difficulties inherent in the foregoing plan of procedure, the most important being the danger of cavity rupture in the course of the apicolysis. This occurred once in one of the early cases in the small series under review, resulting in tuberculous empyema with a fatal outcome. Anybody with experience of apicolysis either in the course of lung resection or thoracoplasty is aware of the difficulty of avoiding cavity rupture in cases where the cavity wall, thinned out and friable, lies firmly adherent to the parietes. Should rupture appear imminent in the case of a cavity adherent to the ribs posteriorly, the lysis from the front should be abandoned for the present until in two weeks time it can be continued from behind after

resection of the posterior ribs. Should cavity rupture actually occur, one should probably remove the upper lobe, if possible, in order to avoid continuing feeding of the infection of the apicolysis space. Should this prove impractical because of the risk, open drainage of the apicolysis space with streptomycin aid seems a possible alternative. It is believed that average care and gentle dissection will prevent cavity rupture if one is alive to the danger and is ready to desist if rupture appears imminent.

The second difficulty concerns the danger of paradoxical respiration owing to the large area of unsupported decostalized chest wall left anteriorly after resection of the upper three ribs and costal cartilages. Support of the infraclavicular region with a large pressure pad hitched to the opposite shoulder and filling of the apicolysis space with normal saline have proved quite satisfactory in overcoming this problem. The pressure pad is kept in position for two weeks after the operation. Experience with resection of the anterior portions of the ribs with and without apicolysis has suggested that there is less trouble with paradoxical movement, when apicolysis is done. This causes complete collapse of the portion of lung underlying the decostalized chest wall, and largely prevents its taking part in respiratory movement. Filling the apicolysis space with saline further helps by supporting the chest wall and compressing the lung.

A further problem arises when a large cavity has been reduced in size by the anterior operation and then shows evidence again of an active inflation mechanism, in a new increase in size, suggesting the need for transpleural drainage. In that the cavity is now more deepseated in its lower position in the more roomy lower chest cavity, and because of its smaller size, its localization for drainage may prove difficult. After unpleasant experience with one such cavity where repeated needle explorations were followed by a fatal hemoptysis, drainage is not now attempted in any such cavity measuring under 4 cm. in diameter. Lung resection is preferred for such a cavity showing a pronounced inflation mechanism or if the inflation element is not marked, further collapse is tried through posterior rib resection.

To counterbalance the aforementioned disadvantages, this procedure seems to have certain advantages over the standard form of thoracoplasty which might be briefly summarized.

1. The patient is operated upon in the supine position for anterior thoracoplasty. This has an advantage for patients with large cavities and profuse sputum in that there is less danger of flooding of the opposite lung than if the patient were lying in the lateral position used in posterior thoracoplasty.

2. For anterior thoracoplasty a short incision is used extending from the midline to the midaxilla at the level of the second rib. The lateral half of the pectoralis major is divided between clamps and the medial half is split. With this approach there is much less blood loss than with the first-stage posterior thoracoplasty.

3. Owing to removal of the wider anterior portions of the upper three ribs and their costal cartilages in their entirety, greater collapse is provided by this operation than by the first-stage posterior operation.

4. Only one operation with no deformity, little or no interference with shoulder function, and minimal reduction of lung function for those patients whose cavities go on to complete obliteration after the anterior operation.

5. Obliteration of the pleural space anteriorly in preparation for transpleural drainage should this prove necessary. Both layers of the pleura become fused with the fibrinous deposit which forms in the apicolysis space postoperatively.

Fig. 1. Fig. 2.

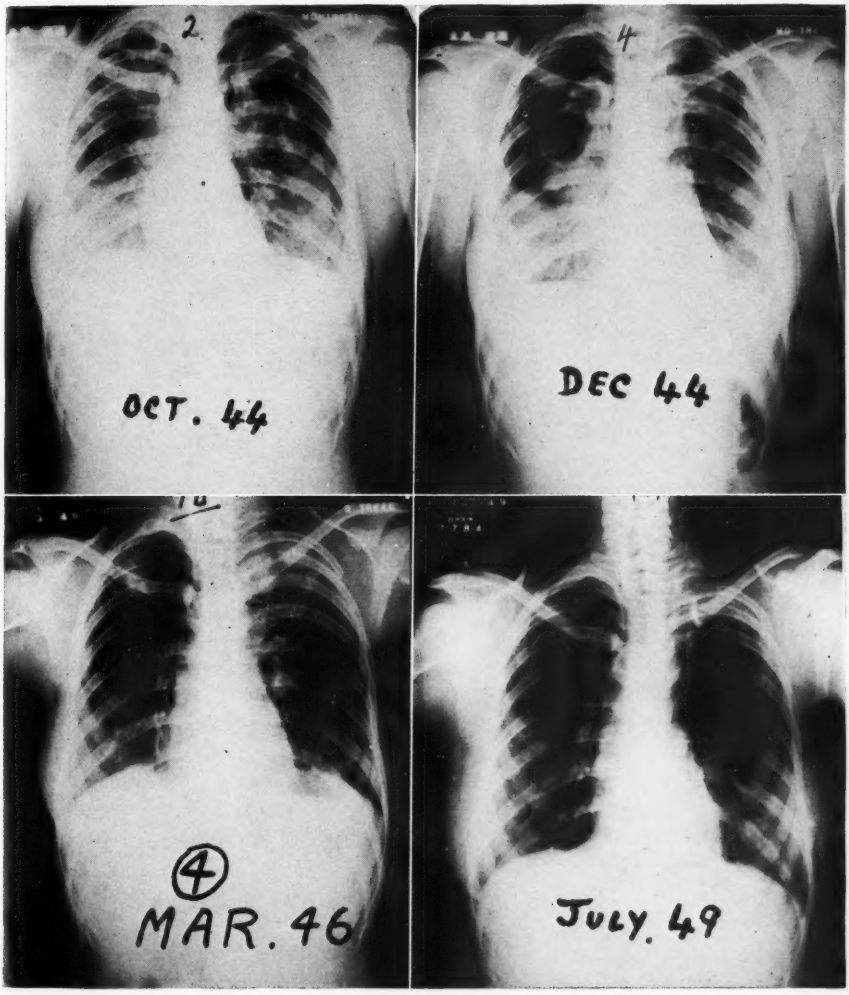

Fig. 3. Fig. 4.

Figs. 1, 2, 3, and 4 (Case 1).—Typical of Group 1 type of cavity behavior.

CASE REPORTS

CASE 1.—Miss J. K., 21 years of age. Onset of pulmonary tuberculosis in June, 1944. Admitted Grace Dart Hospital September, 1944, with 6.5 cm. cavity in both upper zones.

October, 1944, left pneumothorax unsuccessful because of indivisible adhesions, and abandoned. November, 1944, right pneumothorax.

Fig. 5. Fig. 6.

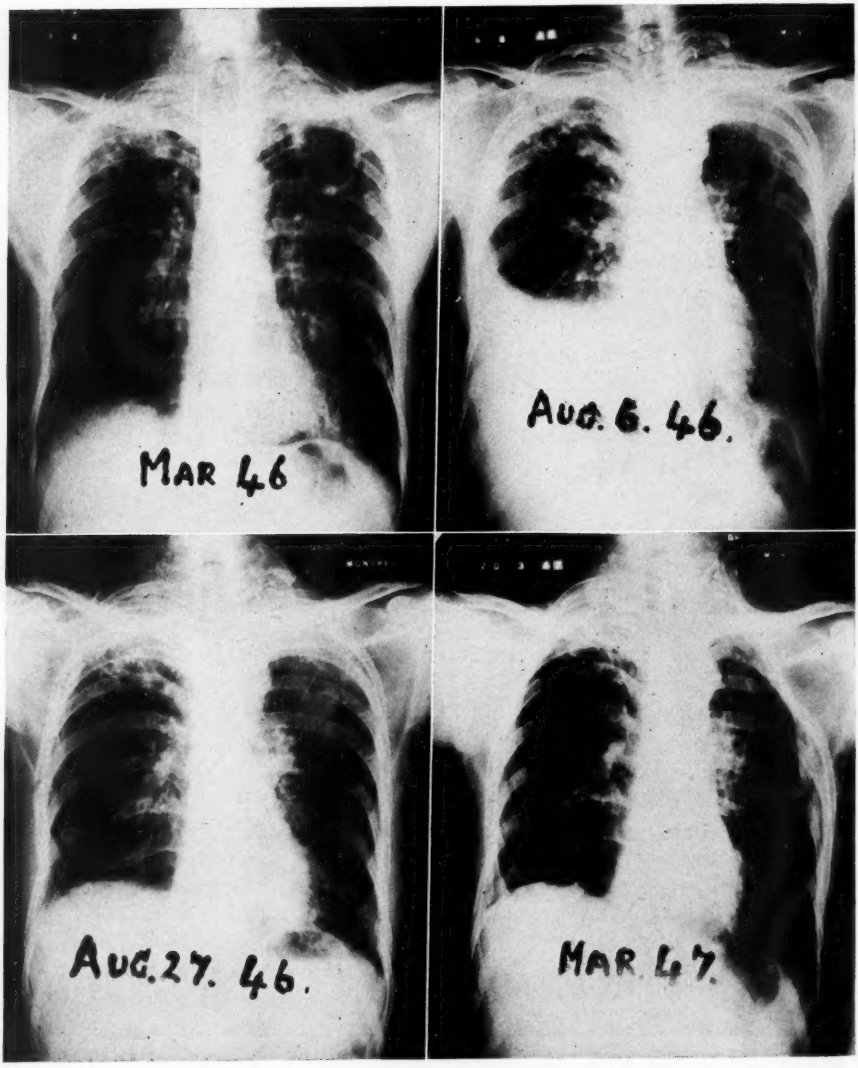

Fig. 7. Fig. 8.

Figs. 5, 6, 7, and 8 (Case 2).—Typical of Group 2 type of cavity behavior.

December, 1944, closed pneumonolysis with division of adhesions and good pneumothorax. The cavity, however, remained as large as before, and the pneumothorax was therefore abandoned. The cavity showed slow reduction in size and gradually disappeared. Cavity pressures were taken in this cavity in August, 1945, after the pneumothorax had become completely absorbed but while the cavity was still a fair size. Pressures were –5, –1 cm. of water. This is interesting in that the behavior of the cavity when it failed to become obliterated under a good pneumothorax suggested that it was in an inflated or positive pressure phase at that

Fig. 9. Fig. 10.

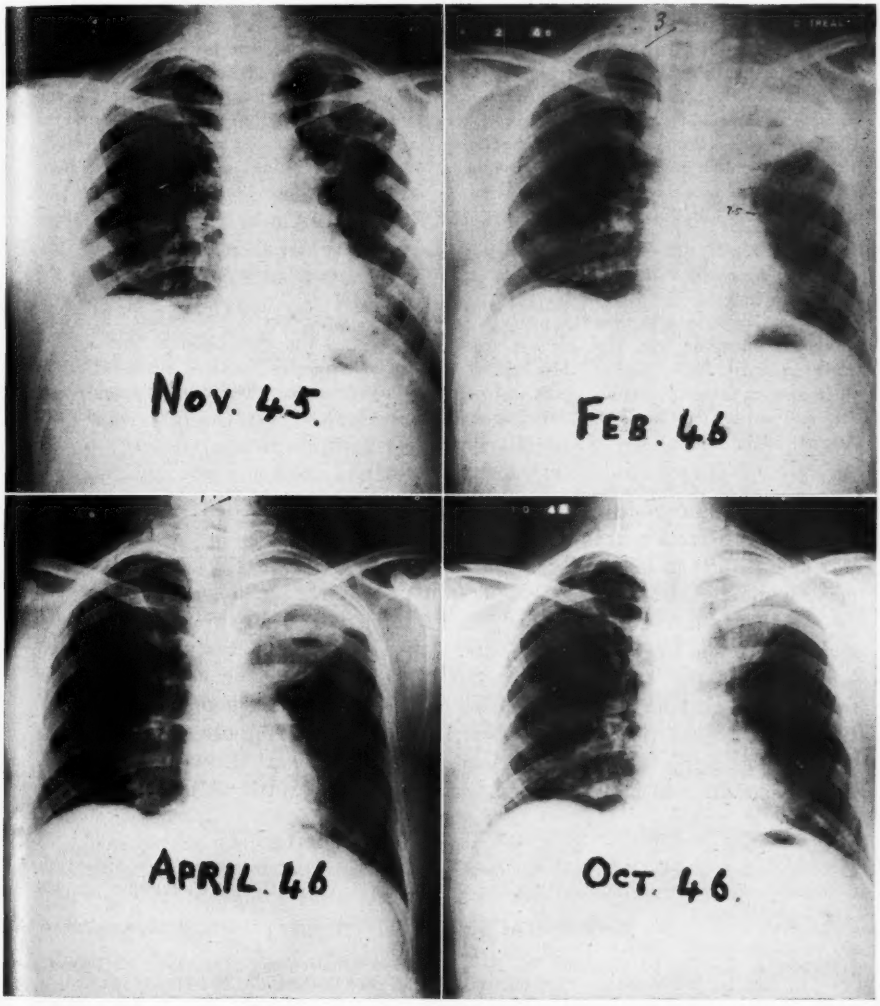

Fig. 11. Fig. 12.

Figs. 9, 10, 11, and 12 (Case 3).—Typical of Group 3 type of cavity behavior.

time. The history of this cavity, going on to obliteration on re-expansion after a period of collapse, suggested a similar course of treatment for the left-sided cavity. Cavity pressures on the left side in March, 1945, were 0, +3 and in December, 1945, were +½, +4.

In January, 1946, left anterior thoracoplasty with apicolysis was done. Sputum became negative in March, 1946, and has remained so. Tomogram has failed to show a cavity in either lung since April, 1946. Discharged in September, 1946. Has since married and has had children.

CASE 2.—Mr. C. R., 44 years of age. Onset of pulmonary tuberculosis in June, 1945. Admitted to Grace Dart Hospital in December, 1945, with 6.5 cm. cavity in left upper zone and 2 cm. cavity in right upper zone.

July 10, 1946, left anterior thoracoplasty with apicolysis.

July 31, 1946, right anterior thoracoplasty with apicolysis. The left-sided cavity was smaller in size after the operation, and then remained unchanged for three weeks after which it rapidly became reduced in size until on Aug. 17, 1946, it measured only 1½ cm. in its longest diameter. It failed to get any smaller over the succeeding three months so that a posterior thoracoplasty with resection of ribs four and five was done in December, 1946. The sputum became negative in October, 1946, and has remained so. Since March, 1947, there has been no conclusive evidence of cavity in the right upper lung.

Discharged in November, 1947. Working as a taxi driver.

CASE 3.—Mr. E. G., 33 years of age. Onset of pulmonary tuberculosis in 1942. Admitted to Grace Dart Hospital in December, 1945. Good chronic with 5.5 cm. cavity in the left upper lung.

Anterior thoracoplasty with apicolysis in February, 1946. Cavity was reduced to 3 cm., after the operation, but rapidly re-enlarged again to 4.5 cm. Transpleural drainage of cavity in April, 1946. Sputum then became negative and remained so until December, 1946, when sputum became positive again. Accordingly posterior thoracoplasty (ribs four, five, six, and seven) in January, 1947. Sputum negative since January, 1947. Discharged August, 1947.

RESULTS

Obviously no valid conclusions as to statistical evaluation of effectiveness can be drawn from a group of sixteen cavities in fifteen patients (one patient was operated upon bilaterally). The results are given in the hope that others may be stimulated by them to adopt similar studies in cavity behavior.

Of the sixteen cavities studied, all but three were 5 cm. or over in diameter and four were in the giant cavity class. It is thought likely that if smaller cavities were included a higher proportion of successful results would have been secured, and that many of the smaller cavities would have gone on to obliteration after the anterior operation without further interference. Smaller cavities are now being treated in this way and will be reported upon, when a sufficient time interval has elapsed to make the results worth while recording.

REFERENCES

1. Eloesser, L.: Blocked Cavities in Pulmonary Tuberculosis, J. THORACIC SURG. 7: 1-22, 1937.
2. Monaldi, V.: Endocavitary Aspiration: Its Practical Applications, Tubercle **28**: 223-228, 1947.
3. Coryllos, P. N.: The Mechanics and Biology of Tuberculous Cavities, Am. Rev. Tubere. **33**: 639-660, 1936.
4. Vineberg, A. M., and Kunstler, W. E.: The Determination and Treatment of Pressure Cavities in Pulmonary Tuberculosis, Surg., Gynec. & Obst. **78**: 245-274, 1944.
5. Hein-Kremer-Schmidt: Kollapstherapie Der Lungentuberkulose, Leipzig, 1938, Georg Thieme.